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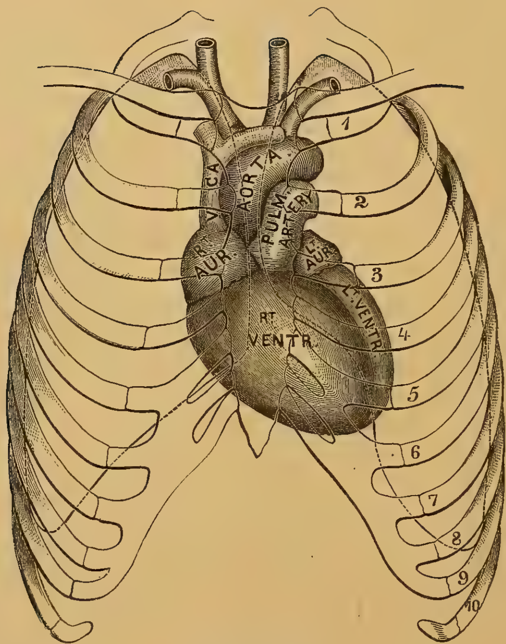
THE HEART AND ITS DISEASES.



FROM DA COSTA.

THE

HEART AND ITS DISEASES,

WITH THEIR

TREATMENT:

INCLUDING THE GOUTY HEART.

BY

J. MILNER FOTHERGILL, M.D.,

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"Without change of opinion there can be no advance in knowledge."

SECOND EDITION (ENTIRELY RE-WRITTEN).

WITH

ILLUSTRATIONS.

PHILADELPHIA:
LINDSAY & BLAKISTON.
1879.

"OBSERVATION has traced back, with fearful fidelity, a long line of formidable and fatal diseases to their pathological parentage in the heart. But that which is constructed of preceding or coincident events, is hitherto less perfect; yet observation has been able to assign to *some* diseases of the heart a sure origin in, and a still continued alliance with diseases of other organs, or of the constitution at large."—PETER MERE LATHAM.

TO

HIS ESTEEMED FRIEND

BALTHAZAR FOSTER, M.D., F.R.C.P.,

IN RECOGNITION OF

HIS CONTRIBUTIONS TO OUR MODERN KNOWLEDGE

OF THE

DISEASES OF THE CIRCULATORY ORGANS,

This Work

IS RESPECTFULLY DEDICATED

BY

THE AUTHOR.

PREFACE TO THE SECOND EDITION.

Conscious of imperfections in the first edition of this work, the author has spared no pains to make this edition worthy of the confidence of the profession. He has striven to describe each form of disease of the heart, not merely as an assemblage of signs and symptoms, but as possessing a natural history; in the belief that such plan will interest practitioner and student alike in the genesis and progress of diseases of the heart; and will furnish indications for treatment, preventive and other, which are not supplied by the plan of regarding diseases of the heart too exclusively from the point of view of the relations of the signs and symptoms found in life to the revelations of the dead-house.

He here gladly acknowledges the aid given him by Dr. Balthazar Foster, who kindly wrote the section on the Sphygmograph, by Mr. Knowsley Thornton, Dr. Pearson Irvine, Mr. Henry Reeves, and Mr. Alban Doran, in different departments. His thanks are also due to Mr. George Evans, the engraver, for the care bestowed upon the tracings. And, finally, he here thanks Dr. H. G. Orlebar for his friendly help in revising the proof-sheets.

23 Somerset Street, Portman Square, W.,
May 24th, 1879.

PREFACE TO THE FIRST EDITION.

WHILE the literature on Heart Disease contains the systematic works of Hope, Stokes, Walshe, and our American *confrère* Flint, it may seem to indicate some lack of diffidence to venture another work on the subject. While, however, we have such work as Claude Bernard, Cyon, Thirry, Von Bezold, Ludwig, and Rutherford are doing in clearing up the important subject of cardiac innervation; Pettigrew investigating the heart's structure and evolution; Brunton vigorously testing the action of remedies upon the heart; Richardson paving the way for a better comprehension of the clinical significance of the heart's objective phenomena; Quain searching into its pathological changes; while Peacock is accumulating a store of information on its malformations, and, finally, George Johnson demonstrating the changes in the arterioles in Bright's disease, and their effect upon the heart, thus giving force to the views of Ludwig and Traube; it is possible that scientific progress may have made room for a newer treatise. In such belief the writer lays this work before the medical public, and if in it many references are made to foreign authorities to the apparent disparagement or neglect of the workers of his own country, it is in no such spirit, however; nor is the work done in England of inferior importance to the pathological researches of the Germans, and especially their minutely exact following out of the consequences of obstructed circulation. The importance of alterations in arterial tension, and the serious re-

sults of venous congestion have been clearly shown by Teuton workers.

The gravity of tricuspid imperfection, as pointed out by Peyton Blakiston in our own country, has especial force given to it by recent research; and the effect of heart failure on the kidneys, and the production of interstitial nephritis therefrom, is now more fairly comprehended.

The importance of Bright's disease in the production of heart disease is now being fully recognized, and the crude opinions of Bright and James are being elaborated by Traube, Ludwig, and George Johnson. The introduction of a systematic chapter on the relation of heart disease and kidney disease to each other is novel, and though the writer's attention has been directed to the subject for years, the present chapter is rather to be regarded as tentative than conclusive—as inviting more attention to the subject than suggesting the exhaustion of it. Finally, the writer must acknowledge the aid derived from the recent treatise on heart disease (Lehrbuch von Herzkrankheiten) of Dr. Th. von Dusch, Professor of Medicine in the University of Heidelberg, who also has permitted the use of his plates, which, along with others from Rindfleisch, will do much to illustrate the subject under discussion.

London, August 6th, 1872.

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THE

HEART AND ITS DISEASES.

CHAPTER I.

THE HEART—ITS EVOLUTION—ITS BLOOD SUPPLY—ITS NERVE SUPPLY—SOME POINTS OF PRACTICAL IMPORTANCE.

WHEN we think about it, we realize what a wonderful thing is that muscular pump within us, throwing the blood from the veins into the arteries; the blood being oxygenized on the way. Asleep or awake, with steady rhythmic stroke the heart keeps on doing its work. Whether at rest in bed, or engaged in active effort, this pump adapts itself and its activity to the needs of the organism—not only without any conscious attention, but in health without even producing consciousness of its action. Such power of adaptation is secured by means of the beautiful nerve adjustments, through which, and by which, the regular rhythmic action of the heart is maintained. Yet the work to be done by the heart varies very much in quietude and in effort. In order to maintain the balance of the circulation, and a steady equilibrium betwixt the bulk of blood in the arteries and in the veins, constant modifications in the heart are necessitated, including variations in the size of the ventricular chambers; and these are rendered possible by the exquisite nerve-balances amidst which the heart swings.

How such a wonderful piece of mechanism as the four-chambered mammalian heart has been evolved; what are the

peculiarities of its own circulation, to meet its varying wants; what the nature of the complex arrangements of its nerve supply, by which its self-regulation is accomplished, are matters calculated to excite the most intense interest. Yet, curiously, both the public who furnish the patients and the medical men who give advice, have to a great extent neglected the normal working of the heart; both, however, feeling keenly the fascination which the diseases of this organ unquestionably possess for all.

The heart in its primitive form is a tube of involuntary muscular fibre, which develops into a pulsatile sac as evolution progresses; this pulsatile sac becomes in time elaborated into the four-chambered heart of the warm-blooded birds and mammals. The following account is abbreviated from the article of the late Dr. John Reid in the "Cyclopædia of Anatomy and Physiology." In the lowest animals, the polypi and actineæ, and a great part of the intestinal worms, the nutritious fluids are transmitted through the tissues without any distinct canals or tubes. In the medusæ vessels are present, but they are unprovided with any pulsatory cavities. In the articulated animals generally, the vessels are still without any pulsatory cavities, but the dorsal vessel has a distinct movement of contraction and relaxation. Various pulsatory dilatations are placed upon the vascular system of the common earthworm; one or two upon the muscular system of the Holothuria; and one in the Talpa Cristata, where the dorsal vessel is reflected upon itself; all of which may be considered as rudimentary hearts. As we rise in the scale of animals we find the pulsatile sac being developed into a two-chambered heart, with the weaker auricle and the more powerful ventricle. Such hearts are found in the Mollusca, and the lowest vertebrata, the fishes; they may drive the blood where a double circulation—a systemic and pulmonary—exists through both the lungs and the system generally. When the batrachians are reached the heart consists of a ventricle and two auricles; one of which receives the blood returning from the respiratory apparatus, the other receives the venous blood of the body. In the serpent the three-chambered heart has a rudimentary septum ventriculorum. In some of the saurians the ventricle is divided into two distinct chambers, which, however, communicate freely with each other. In birds and

mammalia the two circulations, the greater or systemic and the lesser or pulmonic, are distinctly separated from each other.

The development of the heart is a matter so important that I have requested Mr. Alban Doran to write me some further account of it; illustrating the text by engravings from Gegenbauer's "Elements of Comparative Anatomy" (translated by F. Jeffrey Bell, M.A., and revised by Prof. E. Ray Lankester, F.R.S.), which have been furnished me by the courtesy of Messrs. Macmillan & Co.

"Without a sufficient acquaintance with the normal anatomy of the heart, no pathologist can be competent to reason on diseases of that organ. Without some knowledge of the comparative anatomy of the heart, the study of that viscus as seen in our own species, is lifeless, spiritless, and calculated to lead to false conclusions. We shall, on that account, devote a few pages to the consideration of the structure of the heart of the lower animals.

"One of the most interesting facts in comparative anatomy is the frequency, in adult animals of a low type, of the presence of organs bearing the embryonic or infantile character seen in higher forms. Thus the cranium of cartilaginous fishes and the persistent branchial clefts recall conditions well known in the early stage of the mammalian embryo. The ring of thin bone which constitutes the os tympanicum of the ornithorhynchus, and is scarcely better developed in the American monkeys, represents, or rather resembles, the infantile condition of its homologue, the external auditory meatus in man.

"This fact, an unwritten law as we may call it, applies to the heart. In fully developed insects, the centre of the circulation is a rhythmically contractile tube, the dorsal vessel. In the early embryo of man the heart is also a simple tube, which acquires the power of contraction long before it becomes divided into chambers.

"This process of subdivision is seen almost to perfection if the hearts of a fish, an amphibian, a reptile, a bird, and a human subject be laid in a row. The comparative mechanism of the organ is less easy to demonstrate. In some lower forms the heart cannot pulsate with sufficient force to propel blood completely round the vascular circle without peripheral aid. Hence, for ex-

ample, the rhythmically contractile portal vein of *Myxine Glutinosa,* a very low form of lamprey.

"The lowest types of animal life effect their nutrition by osmotic changes through their simple protoplasmic surfaces; but

FIG. 1.

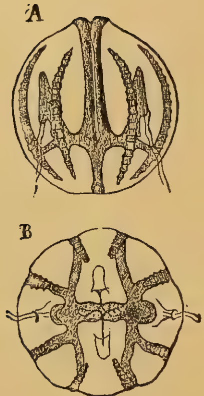


FIG. 2.

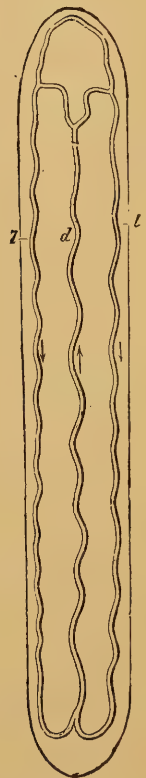


FIG. 3.

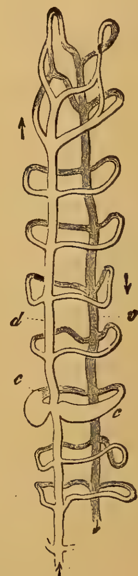


Fig. 1.—The gastro-vascular system of a *Cydippe.* A, lateral view; the mouth turned upwards. B, seen from the oral pole. (After Gegenbauer.)

Fig. 2.—Diagram of the vascular system of the *Nemertina.* *d,* Dorsal longitudinal trunk. *ll,* Lateral vessels. The arrows indicate the direction of the stream of blood. (After Gegenbauer.)

Fig. 3.—Anterior portion of the blood vascular system of *Sænuris variegata.* *d,* Dorsal vessel. *v,* Ventral vessel. *c,* Heart-like enlargement of a transverse anastomosis. (After Gegenbauer.)

singular forms of a true vascular system exist in animals of a very low grade. In the Accalephæ the alimentary canal is modified so

as to form a vascular system as well. The cavity for the reception of food throws out pouches into which the elaborated nutriment enters and is distributed over the system. These pouches sometimes become radiating tubes distributed with beautiful symmetry all over the body (see Fig. 1). This arrangement combines in one the alimentary tract and circulating area. Hence it is termed the 'gastro-vascular system.'

"In *Vermes* a distinct vascular system exists similar to that above mentioned as characteristic of the *Insecta*. There is a dorsal vessel which propels the blood forwards and sometimes communicates with two lateral vessels, which are also pulsatile; the blood returns to the back of the dorsal vessel (Fig. 2). In some of the *Scoleina* the dorsal vessel communicates with a ventral tube by transverse vessels which are pulsatile; and sometimes one or two pairs are dilated, a point of differentiation tending towards the formation of a heart (Fig. 3).

"Among the molluscoida, we find a simple valveless tubular heart in the well-known ascidians. A similar heart exists in some of the entomostraca; but very complicated forms of the organ are to be seen in the higher arthropoda. The heart is often divided transversely into several chambers, which may correspond with the segments of the body; but not invariably so. In the lobster there is a close approach to the vertebrate type of circulatory system, with some remarkable modifications. A true heart exists, surrounded by a pericardium which serves as a venous sinus. The heart gives off four arterial trunks, whose lesser branches end in capillaries which pass into veins. The veins run into a sinus which gives a branch to each gill; the branch, functionally, corresponding to our pulmonary artery. The blood oxygenated in the gills passes into veins which open into the pericardial sinus, and enters the heart through clefts in its walls (Fig. 4).

"The tubular dorsal vessel, representing the heart in insects, is divided by clefts into several chambers, more or less corresponding to the requirements of the body. The blood is driven forwards into the aorta (Fig. 5 *a*), but insects have not the high type of peripheral circulatory apparatus seen in lobsters, for the vital fluid passes from the aorta into lacunæ distributed all over the

body, and returns to the heart through venous spaces in its vicinity.

Fig. 4.

Diagram of the circulatory system of a lobster. (After Gegenbauer.) *o*, Eye. *a e*, Lateral antennæ. *a i*, Mesial antennæ. *b r*, Branchiæ. *c*, Heart. *p c*, Pericardium. *a o*, Median anterior aorta. *a a*, Hepatic artery. *a p*, Posterior artery of body. *a*, Trunk of the ventral artery. *a v*, Anterior ventral artery. *v*, Ventral venous sinus. *v br*, Branchial veins. The arrows indicate the direction of the current of blood.

"In the myriapoda the long heart, or dorsal vessel, is very distinctly divided into segments corresponding to the extreme seg-

mentation of these many-footed animals. But it is most important to remember that there are valves between the chambers of the

Fig. 5.

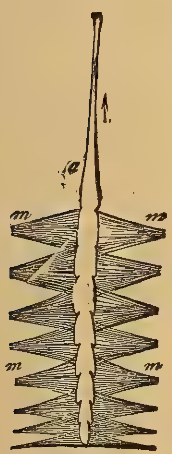


Fig. 6.

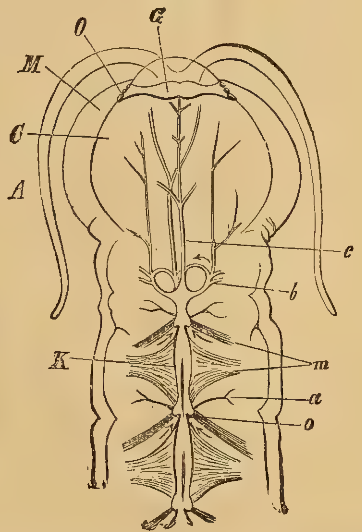


Fig. 5.—Heart of Cockchafer. (After Gegenbauer.) *a*, Artery arising from the most anterior chamber. *m*, Alæ cordis. (After Burmeister.)

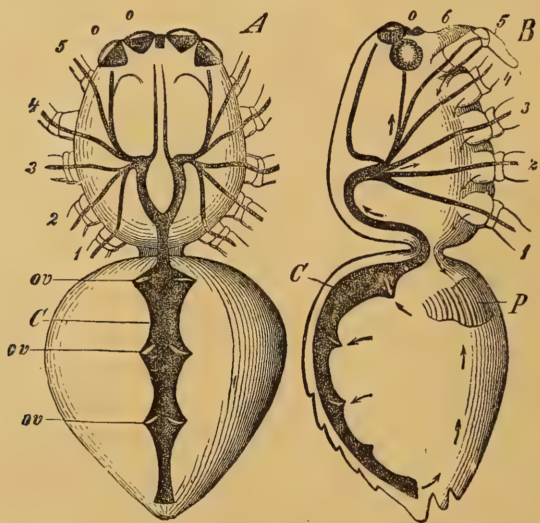
Fig. 6.—Head and two segments of the body of *Scolopendra*. (After Gegenbauer.) *K*, Chamber of the heart. *m*, Alæ cordis. *e*, Venous ostia. *a*, Lateral arteries. *b*, Arterial arches. *c*, Cephalic artery. (After Newport.)

heart; and as the valves cover the orifices of veins, it is clear that the segmentation of the heart has some important functional, and not a purely morphological, signification. The scorpions much resemble the lobster in their circulatory apparatus. The spiders have the more primitive arrangement seen in the insects, but the heart is ventral, not dorsal, and gives off arteries to the eyes and limbs. The remainder of the track of the blood-current is lacunar, and the blood returns to the heart through distinct clefts, or 'venous ostia.'

"In the Mollusca the heart attains a high degree of development. The dorsal trunk is dilated into a ventricle, and gives off branches which are also frequently dilated into auricles. The following diagram (Fig. 8), from Gegenbauer, demonstrates the transitional conditions of the heart in the Mollusca, the organ

having in the lower forms the character of the dorsal vessel with transverse branches seen in vermes (Fig. 2); whilst in the cuttle-fish and snail we see a very near approach to the vertebrate heart.

FIG. 7.



Circulatory organs of Lycosa (Spider). (After Gegenbaur.) *A*, seen from above; *B*, from the side. *o*, Eyes. 1, 2, 3, 4, 5, 6. Appendages. *P*, Lungs. *C*, Heart. *o v*, Venous ostia of the heart. The arrows indicate the direction of the blood. (After Clarapède.)

"In the vertebrate embryo the heart is at first a mass of cells, soon becoming tubular. It remains a simple tube in that extremely primitive animal, the *Amphioxus*. But this little fish has a complete vascular system, not lacunar, as in insects.* There is even a rudimentary portal system, although the liver is a mere diverticulum from the intestine.

"The tube passes anteriorly into an aortic trunk, and receives from behind the omphalomesaraic veins from the umbilical vesicle. This primitive heart in the vertebrates above *Amphioxus* becomes bent on itself. The middle portion of the tube at the angle of the bend bulges and constitutes a ventricle. The

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* Most of its veins are contractile.

extremity receiving the veins becomes an auricle. The opposite end dilates below the point where it gives off the aortic trunk. This is termed the bulbus arteriosus. In the fish the heart remains in this condition throughout adult life. Fig. 9 represents

FIG. 8.

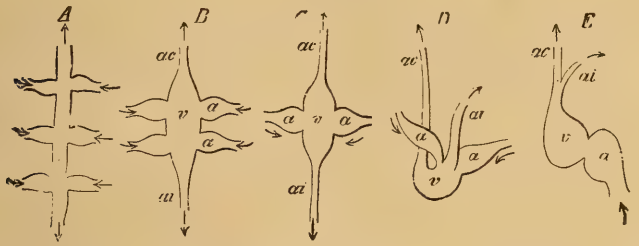


Diagram to show the relations of the circulatory centres in the Mollusca. (After Gegenbauer.) *A*, Part of the dorsal vascular trunk and transverse trunks of the Worm. *B*, Heart and auricles of a Nautilus; *C*, Of a Lamellibranch, or of Loligo; *D*, Of Octopus. *E*, Heart and auricle of a Gasteropod. *v*, Ventricle. *a*, Auricle. *a c*, Arteria cephalica. *a i*, Arteria abdominalis.

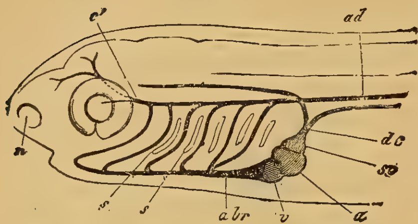
the heart in an osseous fish. The single ventricle is prolonged towards the bulbus arteriosus, the prolongation being termed the *conus arteriosus*. This conus is provided in the sharks with a regular flight or row of semilunar valves, developed (as are all the cardiac valves of man) as processes of the endocardium. Two beautiful preparations in the museum of the College of Surgeons* illustrate the chambers and multivalvular conus arteriosus in *Lamna* and in the great basking-shark (*Selache*).

"In these vertebrates venous blood is returned to the heart by the sinus venosus, which conducts the fluid to the auricle, thence to the ventricle, which distributes it over the branchial arteries. These vessels are branches of the aorta supplied to the gills, and permanent representatives of the temporary branchial arches of higher animals. The blood is oxygenated in the capillaries of the gills, and is returned by branchial veins into the aorta, and thence passes into the systemic circulation (Fig. 9). This also represents how the branchial arteries are placed in relation to the aorta. All are originally developed from the primitive arterial trunk at one extremity of the heart.

* Physiological Series, 911 D, 911 G.

"In the mammalian embryo, after the formation of the three divisions persistent in most fishes, septa form between the auricles, and also between the ventricles, and the primitive arterial trunk and bulb also undergo subdivision into the roots of the aorta and pulmonary artery. In man the conus arteriosus remains as a funnel-like prolongation of the right ventricle towards the pulmonary artery, free from columnæ carneæ. Every step towards this high stage of development may be observed in adult vertebrates between pisces and mammalia.

FIG. 9.



Head of an embryonic Teleostean (diagrammatic). (After Gegenbauer.) *a*, Auricle. *v*, Ventricle. *a br*, Branchial artery. *c*, Carotid. *a d*, Aorta. *s*, Branchial clefts. *n*, Nasal pit. *s v*, Sinus venosus. *d c*, Ductus Cuvieri.

"The auricle of the singular *Lepidosiren*, which is provided with both gills and lungs, is partially divided into two chambers by muscular bands, and a similar arrangement exists in the ventricle, extending into the arterial bulb. In the tadpole there is, as in fishes, a single auricle and a single ventricle, and the relation of the branchial arteries to the aorta is the same. But in the adult frog and other amphibia the auricle has become divided into two chambers; the right receiving venous blood, the left blood from the lungs.

"In most reptiles the heart has two auricles and one ventricle (Figs. 10 and 11), as in the frog. To avoid any dangerous admixture of venous with oxygenated blood, a muscular process projects into the ventricular cavity, dividing the streams almost as effectually as if a true septum existed.

"In crocodiles and birds the ventricle is divided into two chambers, as well as the auricle. In this respect these vertebrates resemble the mammalia; but differ in the ultimate development of the aortic arches—a subject too complicated for discussion in these pages. The nature of the arterial and auriculo-ventricular valves varies much in different orders of the lower classes of the vertebrata.

"The most remarkable modification in the heart amongst the mammalia, is seen in the *Sirenia,* or manatees and dugongs, where

Fig. 10.

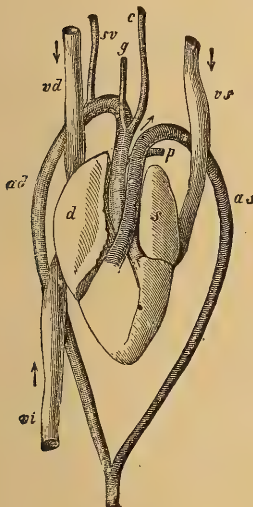


Fig. 11.

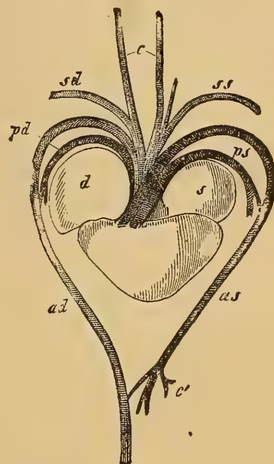


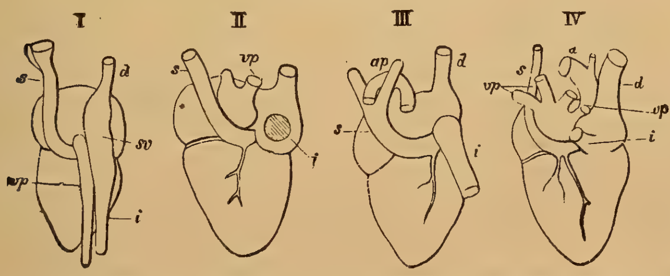
Fig. 10.—Heart and arteries of an Ophidian (Boa). *d*, Right; *s*, left auricle. *c*, Carotid. *a d*, Right; *a s*, left aortic arch. *p*, Pulmonary artery.

Fig. 11.—Heart and arterial trunks of a Chelonia (Chelydra, Turtle). *d*, Right; *s*, left auricle. *c*, Carotid. *a d*, Right; *a s*, left aortic arch. *p*, Pulmonary artery.

the septum ventriculorum is so deeply grooved as to cause the right ventricle to be half separated from the left.

"The different forms of the heart of the vertebrata are given in the following plate:

Fig. 12.



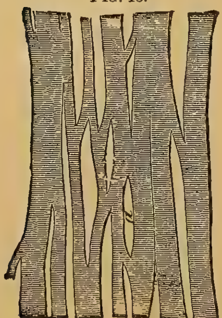
I. Reptile. II. Bird. III. Marsupial. IV. Pig. They are seen from behind in order to show the relations of the great venous trunks to the heart. *i*, Vena cava inferior. *s*, Vena cava superior sinistra. *d*, Vena cava superior dextra. *a p*, Pulmonary artery. *a*, Aorta. *s v*, Sinus venosus.

"Any account of the general anatomy of the human heart would be out of place here; but the foregoing account of the development of the heart may be interesting to readers who wish to learn something of the subject, and have not a convenient text-book at hand."

The heart consists, then, first of a tube of involuntary muscular fibre, which is gradually drawn together and shortened until a pulsatile sac is formed of several layers of muscular fibres; until, as B. W. Richardson says, it becomes "a coiled spring." These different muscular fibres are intricately interlaced, some fibres being common to both sides of the heart, while others are confined to one side. Thus the heart was described by Winslow as consisting of two muscles enveloped in a third. These fibres have been unravelled with marvellous patience by the late H. Searle, and by Dr. Pettigrew. The latter has shown that the fully developed heart consists of seven layers of fibres; the innermost passing spirally from base to apex, the centre layer being almost circular, while the outermost layer again passes spirally from apex to base. By such an arrangement the blood is effectually squeezed out of the cavities at each muscular contraction.

The muscular structure of the heart consists of involuntary muscular fibre; but these fibres have lost their primitive fusiform shape, and have got the cross-markings of voluntary muscular fibres. The muscular fibre of the heart is reticulated.

Fig. 13.



Muscular Fibre of Heart. (From Rindfleisch.)

Within the sarcolemma of each muscular bundle are rudimentary elements of fibres, from which, it is believed, the new fibres are developed in hypertrophy (or hyperplasia), and from which new fibrillæ are furnished when the original fibres are destroyed by the acute myositis of pyrexia, and notably of relapsing fever.

The heart is divided into two halves, of unequal size and power in most animals; but in the diving mammals the right heart is largely developed, and in the dugong the heart presents the appearance of a double Barcelona nut. Each half has

(1), the first chamber, the auricle, a comparatively thin-walled chamber; and (2), the ventricle, whose walls are thicker. The systole, or contraction commences first in the auricle, and then passes on swiftly to the ventricle. It is a true vermicular action, though a rapid one. The action passes from one layer of fibres to another, as first shown by Schiff and confirmed by Valentin. The action is the same as that of the intestine, only the muscular fibre-tubing is coiled upon itself, and the contraction is, consequently, nearly synchronous. The blood wells into the empty chambers from the full veins behind, until the pressure is alike in the veins and the heart chambers; then the auricle contracts towards the ventricle, the ventricle is next excited to contract; and the ventricular contraction closing the auriculo-ventricular valves, the fluid contents are expelled by the open orifice into the artery in front. Such is the cardiac cycle. Dr. Michael Foster describes the cycle more completely. He says: "When the chest of a mammal is opened and artificial respiration kept up, a complete beat of the whole heart, or cardiac cycle, is seen to take place as follows:

"The great veins, inferior and superior venæ cavæ and pulmonary veins, are seen while full of blood to contract in the neighborhood of the heart; the contraction runs in a peristaltic wave towards the auricles, increasing in intensity as it goes. Arrived at the auricles, which are then full of blood, the wave suddenly spreads, at a rate too rapid to be fairly judged by the eye, over the whole of those organs, which, accordingly, contract with a sudden sharp systole.

"In the systole, the walls of the auricle press towards the auriculo-ventricular orifices, and the auricular appendages are drawn inwards, becoming smaller and paler. During the auricular systole, the ventricles may be seen to become more and more turgid. Then follows, as it were immediately, the ventricular systole, during which the ventricles become shorter and thicker. Held between the fingers they are felt to become tense and hard. As the systole progresses, the aortic and pulmonary arteries are seen to expand and elongate, and the heart to twist slightly on its long axis, so that, while the base is fixed by the great arteries, the apex moves from the left and behind to the front and right;

hence, more of the left ventricle becomes displayed. As the systole gives way to the succeeding pause or diastole, the ventricles flatten and elongate, the aorta and pulmonary artery contract and shorten, the heart turns back towards the left, and thus the cycle is completed."

In connection with this subject, it may be well to consider another point, viz., the rapidity of the blood flow. This is greatest at the aortic orifice, is rapid along the arteries as they spread out and the area enlarges,—the greater the area the slower the flow. The flow increases as it passes from the venules to the veins, being greatest in the vena cava, where the area has again become small. The flow is equal, therefore, and as much blood enters the right auricle as issues from the left ventricle at each stroke. The flow in the right heart is slower, therefore its ostia are wider. "If it be true, as it undoubtedly is, that equal volumes of blood must pass synchronously through the pulmonic and aortic openings, and that the two ventricles exert very unequal pressures upon the equal columns of blood in systole, it must follow, if the velocities are unequal, that equal times of afflux can only be maintained by the areas of the aortic and pulmonic orifices being inversely as the velocities of the streams which traverse them." (Herbert Davies.)

The ostia of the right heart are larger than those of the left heart, while the auriculo-ventricular orifices bear a regular proportion to the arterial ostia. The aortic orifice is the smallest, the pressure exerted on it by the powerful left ventricle is greater than that exerted on the pulmonary orifice, and at that point the flow is the most rapid. The vascular area spreads out from the aortic orifice to the capillaries; and gathers together again to it through the right and left hearts. The quantity of blood which is driven from each ventricle at each systole in a full-grown man of average size and weight, is 180 grams—six ounces. (M. Foster.)

As might be expected, the thickness of the muscular walls on each side is in proportion to the work to be performed. The difficulties to be overcome by the right heart are comparatively small. It has to drive the blood through the large capillaries of the pulmonic circulation into the left chambers of the heart. The

left heart has to overcome the elasticity of the systemic circulation, and consequently is much thicker. Various calculations have been made as to the relative thickness of the walls of the two ventricles; but they vary from that of Laennec, who thought the left ventricle as rather more than two to one the right ventricle; to that of Cruveilhier, who thought the proportions as high as five to one. Bizot made the most elaborate measurements of the heart in one hundred and fifty-seven individuals of all ages, but they are too minute and lengthy for quotation. Hayden gives as a general estimate of the thickness of the various portions of the heart-walls, the left ventricle seven lines, the right ventricle two and a half. The thickness of the interventricular septum, eleven lines. The mean thickness of the left auricle is one and a half lines, of the right auricle one line. The weight of the heart varies with age, and increases as life progresses. It is estimated at eight ounces for the adult female, and nine ounces for the average male.

The muscular fibres are arranged in connection with tendinous rings which form the different ostia of the heart. The arterial tendinous rings are continuous with the middle arterial coats; the fibres contract towards the ostia, and so propel the blood forwards. In order to prevent regurgitation of the blood, each ostium is protected by valves; the arterial orifices have each three semilunar valves, which lie close to the arterial wall when the blood is rushing through the ostia in the ventricular systole; but which approximate each other (by virtue of elastic fibres at the base of each cusp, according to some authorities) in the aortic rebound, and are tightly driven together, thereby preventing effectually all reflux. The auriculo-ventricular valves consist of curtains, which are prevented from being washed backwards by tendinous cords attached to them, the tendinous cords being also attached to small muscular masses, the musculi papillares, springing from the walls of the heart. On the ventricular contraction, these curtains are approximated by the force of the regurgitant current, and prevent reflux; without these chordæ tendineæ the auriculo-ventricular valves would be washed into the auricles at every ventricular systole. The musculi papillares contract synchronously with the ventricles and draw the valve curtains inwards on the systole. In

the bird, the auriculo-ventricular valves are muscular and take part in the general contraction of the muscular walls. This is especially the case in diving birds; while in the ostrich tribe the valve arrangements resemble those of mammals. The practical profit to be derived from further consideration of the anatomical details of the heart do not seem to be such as to warrant their consideration here. The right ventricle is applied around one side of the left ventricle.

The nutrition of this wonderful muscular and tendinous mass is secured by two arteries which spring from the base of the aorta, the coronary arteries. The right coronary artery arises from the anterior and right sinus; the left from the anterior and left sinus (Hayden). Sibson says there are two aortic posterior flaps, from one of which the left coronary artery springs. The right coronary artery descends between the root of the aorta and the right auricular appendix, and supplies the right ventricle. The left coronary artery descends obliquely to the left, between the pulmonary artery and appendix of the left auricle (Hayden). Anastomoses exist between the branches of the right and left coronary arteries; but, according to Hyrtl, they are too small to admit of the passage of ordinary injection material. The blood gathers together again into veins, one of which is distinctly larger than the others, and has upon it a distinct sinus, which Reid has found to be muscular in structure in various animals; these veins open into the right auricle. In perforating the muscular wall from without inwards, these veins pass obliquely through the wall, so that when the auricle is distended, reflux into them is prevented; just as reflux from the bladder into the ureters is prevented. Imperfect valves are described, especially at the orifice of the large coronary vein, further preventing the entrance of venous blood from the right auricle into the coronary circulation, when the right heart is distended.

There are certain points of practical interest connected with the coronary circulation. In the first place, though the coronary arteries spring from the sinuses of Valsalva, just above the line of the aortic cusps, when laid back by the ventricular systole; still, the systole of the ventricle does not fill these arteries. The coronary arteries are filled by the aortic rebound, from the blood then

eddying in the sinuses of Valsalva. During the cardiac systole, the blood is wrung out of the small vessels of the coronary circulation, by the twisting action of the ventricular contraction; when the systole is over the coronary arteries and their branches are empty, ready to receive the arterial blood driven into them by the aortic recoil.

The coronary circulation is protected from any regurgitation of venous blood into it by the mode of perforation of the coronary veins and the imperfect valves, otherwise the heart would be placed under great disadvantages; it would be gorged with venous blood, which would tend to directly paralyze it from the presence of carbonic acid, while it would be negatively harmful from its obstructing the ready flow of arterial blood through the coronary circulation during the heart's diastole.

During the diastole, the heart gets its brief sleep and its supply of pabulum for tissue nutrition. In its incessant round of labor, no long repose is compatible with the continuation of the existence of the organism. It must not, however, be supposed that the actual amount of sleep enjoyed by the heart is small; that would be incompatible with its active function. The diastole occupies three-fifths of the time of the whole cardiac cycle—*i. e.*, on the aggregate, about fifteen out of the twenty-four hours. In the first edition the time is differently stated; here the time of the diastole as given by M. Foster is followed. In disease, when the pulse becomes rapid, the number of cardiac contractions in the minute is increased, and this increase takes place at the expense of the general diastolic pause—*i. e.*, the duration of each systole is but little affected; but there are so many more of them in the minute, and each diastole is shorter, and the aggregate diastole is diminished by the increased number of systoles in the minute; consequently the heart's sleep is much diminished. It has not only to work much longer in proportion to its rest; but its working time is increased, while its sleeping time is distinctly diminished. On the other hand, Balthazar Foster has found that when the heart's action is slowed by digitalis, the slowing is procured by the lengthening of the diastole. Consequently, we can see how the heart's sleep, alike in its nervous ganglia and its mus-

cular fibres, may be influenced by the administration of therapeutic agents.

For the following account of the anatomy of the cardiac nerves I am indebted to H. A. Reeves:

"The cardiac nerves come, as is well known, from the pneumogastric and sympathetic. The former nerve gives off branches to the heart from its cervical as well as from its thoracic portions. The upper cardiac nerves are minute filaments, and usually join the cardiac nerves of the sympathetic; the lower is a single branch, and is generally given off just before the nerve enters the thorax. There is a difference in the arrangement of this latter nerve on the two sides. On the right side it rests on the side of the innominate artery, and joins a cardiac nerve of the sympathetic (occasionally of the recurrent laryngeal), to the deep cardiac plexus. The left branch crosses the aortic arch to join the superficial plexus. The thoracic cardiac branches are given off from the pneumogastric of the right side near the trachea, and a few come from the first part of the recurrent laryngeal; they rest on the trachea and go to the deep cardiac plexus. On the left side the left recurrent laryngeal gives off corresponding nerves.

"There are three cardiac branches of the sympathetic, which arise respectively from the three cervical ganglia of that nerve trunk, and there are differences in the arrangement on the two sides of the neck. The *right* upper cardiac nerve comes from two or more filaments of the upper cervical ganglia, and occasionally there is an offshoot from the cord joining the upper and middle cervical ganglia. It is situated behind the carotid sheath, and in front of the inferior thyroid artery and recurrent laryngeal nerve. In the thorax it passes sometimes in front and sometimes behind the subclavian artery, and runs along the innominate artery to the back of the aortic arch to join the deep cardiac plexus. Some twigs are given off to the anterior aspect of the arch of the aorta, to the superficial cardiac plexus. This nerve communicates in its course with the external and recurrent laryngeal nerves, and with other branches of the sympathetic and pneumogastric. The *left* upper cardiac nerve differs from its fellow in the *thoracic* part of its course, where it runs on the left carotid to the aortic arch. It may pass in front of the arch to the superficial plexus,

or behind it to the deep cardiac plexus. The *middle* cardiac nerve of the right side is placed behind the carotid sheath, and either before or behind the subclavian artery. It lies on the trachea in the thorax, and is joined by twigs from the recurrent laryngeal, and ends on the right of the deep plexus. Its nuchal communications are with the upper cardiac of the sympathetic and the recurrent of the pneumogastric.

"The *left* middle cardiac nerve passes into the chest between the left carotid and subclavian to the left side of the deep plexus.

"The *lower* cardiac nerve comes from the lower cervical ganglion, or from the first thoracic. On the *right* side it passes behind the subclavian artery, and ends in the deep plexus. It anastomoses with the recurrent laryngeal and middle cardiac nerves. On the *left* side it often joins the middle cardiac, and the resulting cord ends in the deep plexus. Sometimes the deep plexus is replaced by a ganglion called Wrisberg's ganglion, and from it or the plexus filaments are given off and accompany the heart arteries. Besides these, the heart has three special ganglia, and some smaller unnamed ones. The first is situated near the entrance of the inferior vena cava, and is called Remak's ganglion; the second is near the left auriculo-ventricular valves, and is named the ganglion of Bidder; and the third is in the wall of the right auricle, and is Ludwig's ganglion. Dr. Robert Lee, in 1864, described a rich system of ganglionic nerves, which are partly subpericardial, and partly in the muscular walls of the heart.

"There is much yet to be done in the anatomy of the cardiac nerves."

The subject of the innervation of the heart is a very difficult one; and what is put forward here is merely that about which all are agreed, rather than any attempt at an exhaustive handling of the matter. Such consideration will, however, help us much to a correct comprehension on some matters of much practical moment, both clinically and therapeutically.

In the first place the heart possesses ganglia of its own, rhythmically discharging centres, which can carry on the movements of the heart for a considerable time after its removal from the body. (This, of course, applies only to cold-blooded animals, on

whom alone such experimentation is possible.) "We shall take for granted that the principal laws of the heart's beat, which are shown by the frog's heart, also hold good for those of higher animals." (M. Foster.) These lie in the sinus, in the auricular septum and walls, and in the auriculo-ventricular groove, though some have been found in the heart itself. These ganglia belong to the primitive pulsatile sac, and remain in the highest form of heart.

These ganglia receive the sense of distension and give off efferent impulses to the muscle to contract. Their afferent branches are in association with the cavity or inner surface of the chambers; the efferent branches with the muscular fibrillæ. When the heart is removed from the body, and fluid is driven into the cardiac cavities with abnormal force, more rapid and more energetic contractions are set up. This is the primitive nerve-balance of the heart. Upon this primitive base some very complex nerve arrangements are superimposed, so that the heart swings in the most beautiful nerve adjustments.

In intimate connection with these nerve ganglia is the vagus nerve. The brothers Weber in 1846 showed that irritation of the vagus nerve would slow the action of the heart, while greater irritation would bring the heart to a standstill. Section of the vagi leads to tumultuous action of the heart. The right vagus exerts a more powerful influence on the heart than the left. Since the original observations of the Webers, the vagus has received a great deal of attention. It is a rope of several strands, mostly efferent from above downwards. It contains (1), inhibitory fibres; (2), reflex inhibitory fibres; and (3), afferent fibres to the vasomotor centre, the sensory nerve of the heart of Ludwig.

The direct inhibitory fibres control the ganglia of the heart, and by their restraining action hold back the explosion generated in the ganglia, until a synchronous contraction is secured. (Hence when the vagi are cut, the contractions are tumultuous.) The famous Czermak, by pressing the vagus in the neck against an osseous prominence, could arrest the action of his heart. Several individuals have been able to exercise a decided control over their hearts by an effort of will. It is said that Fontana could slow and accelerate the action of his heart at will. This was well seen

in the notable case of Colonel Townsend. "A few days before his death he could expire when he pleased, and by an effort he could come to life again. Before making the experiment on the day of his death his pulse was distinct, though small and thready, and his heart had the usual beating. He composed himself and lay on his back in a still posture for some time. His pulse sank gradually, till at last it could not be felt by the nicest touch; not the least motion could be felt in the heart, nor the least soil of breath perceived on a bright mirror held to his mouth; not the least symptom of life could be perceived. He continued thus for half an hour; the motion of the heart was then observed gradually to return, and he recovered again, executed some business, and died the same evening." (Wardrop, "Diseases of the Heart," p. 87.) There are, then, central inhibitory fibres more or less under the control of the will. By these the cool individual calms his heart under excitement, as seen in the practiced rifleshot.

Then there are reflex inhibitory centres, those by which the heart is brought to a standstill in diastole, as when the intestine is struck sharply with a knife (Goltz). Such, probably, is the explanation of the instant death which follows a severe blow on the pit of the stomach.

Through these inhibitory fibres, fainting from severe emotion, injury, or shock is brought about.

The third strand consists of fibres which run from the heart to the medulla. These fibres have been called the sensory nerve of the heart, or vaso-inhibitory nerve (Ludwig). When the heart is unduly distended, these fibres are thrown into action, and inhibit the vasomotor centre, so that the peripheral arterioles are dilated, and the blood pressure in the arteries lowered. The opposition to the outward flow from the heart is thus lessened, and the muscular walls can more easily contract, and recover themselves from the state of overdistension.

In addition to these actions, Eichhorst has found that section of the vagi in birds leads to fatty degeneration of the heart fibres; while section of the vagi in rabbits and dogs is followed by changes in the muscular fibres, but death follows too soon to admit of this being shown to be fatty degeneration. He does not put forward any hypothesis as to the *modus operandi* of what he

terms the trophic nerve fibres of the vagus; but our acquaintance with the contraction of the bloodvessels of the stomach when the vagi are cut, renders it not improbable that the sympathetic fibres, no longer antagonized by the cerebro-spinal fibres, contract the bloodvessels and impaired nutrition results.

The practical bearings of the innervation of the heart are these. When a powerful ventricle is pumping blood forcibly into the arterial system, the blood pressure is high, and the roots of the vagus are flooded with blood; the inhibitory fibres are thrown into action, and the cardiac ganglia are restrained and the heart beats slowly. Without such inhibitory arrangements apoplexy would be very common. The powerful ventricle would constantly be bursting the arteries, especially of the brain, if the inhibitory fibres did not hold back the contraction of the ventricle, and so allow of the blood in the arteries escaping through the arterioles before the next ventricleful of blood is thrown into the arteries. The inhibitory fibres of the vagus thus protect the arteries. The pulse of high blood pressure is slow, then—but it is powerful.

The reflex inhibitory fibres are those which cause syncope under shock, injury, or emotion. Such syncope leads directly to unconsciousness, and by this the brain is saved from the full force of the shock; which, if it fell upon a brain not anæsthetized by unconsciousness would do severe and serious injury. Syncope then saves the brain from the full force of shocks, either material or emotional.

The use of the accelerator fibres—to be mentioned shortly—is shrouded in mystery. It is possible to speculate that they are useful when the heart has been seriously depressed, and they may counteract the influence of the inhibitory fibres when their action is excessive. They are not exhausted by excessive stimulation, "they may be subjected repeatedly to the action of strong currents without appearing to suffer" (M. Foster). Whatever their use, they are clearly very important nerve fibres.

The function of the sensory, or vaso-inhibitory nerve, is a very important one. It protects the heart itself from paralytic overdistension. When the cardiac chambers are overdistended, the sense of overdistension is conveyed by afferent fibres of the car-

diac ganglia to these centres, and more rapid and efficient discharges take place, resulting in more rapid and energetic muscular contractions. Not only this, but the sensory nerve of the heart being the vaso-inhibitory nerve, the peripheral arterioles are dilated and the blood-flow out of the arteries is rendered easier. By this readier outflow, with lowered blood pressure in the arteries, and therefore less obstruction to overcome in systole, the more energetic contractions of the heart are assisted, when the cardiac chambers are overdistended.

There are, however, other nerve fibres passing from the medulla to the heart, which are termed the accelerator nerves, and which quicken the beat of the heart. "The accelerator or motor nerves of the heart pass from the medulla down the spinal cord, and reach the heart through the last cervical and first dorsal ganglia of the sympathetic nerve." (Ferrier.) These nerve fibres, according to M. Foster, "pass from the cervical spinal cord, along the nerve accompanying the vertebral artery, and reach the heart through the last cervical and first thoracic ganglia." Through the accelerator nerves the heart's action may be quickened, even after the division of both vagi, by direct stimulation of the cervical spinal cord. These nerves act slowly and are in no way antagonistic to the vagi. Stimulation of these nerves increases the number of the heart beats; but what is gained in rate is lost in force, for the blood pressure may remain the same, or even be diminished. "We know nothing of the action of these nerves in the economy, unless we suppose they are of use in cases where it is desirable the heart's beat should be quickened without any change in the pressure." (M. Foster.) As to the destination of these accelerator nerves, "They end probably in the intracardiac ganglia, reinforcing, in some manner unknown, their automatic activity. Stannius has shown, that in the heart of the frog, at all events, there are ganglionic centres having independent functions, one class acting simply as *reflex* centres; a second, called *accelerating* centres, which seem to quicken the cardiac movements; whilst a third, named *inhibitory* centres, retard or arrest these movements. Thus rhythm may depend on the first alone, but the amplitude, and the time of the rhythmic contraction, may be regulated by other centres." (McKendrick.) Thus it would

seem, that the swing of the cardiac pendulum is secured by more complex and subtle nerve arrangements than the primitive nerve-balance—the reflex centres.

We see, then, that the primitive cardiac ganglia with their simple afferent and efferent fibres, are controlled and materially aided by the other and more complex nerve arrangements of the heart; so that in quietude and in effort, the heart's action is adapted to circumstances; that, indeed, the heart swings in a very exquisite and complex set of balances.

A word as to the action which goes on in these cardiac ganglia may not be out of place here. These ganglia, and the respiratory centres in the medulla, are motor centres which discharge at intervals rhythmically. There seems a certain accumulation of energy in the cells that constitute these discharging centres, which explodes rhythmically. The basis of action is the decomposition set up in these nerve cells, which contain compounds of most elaborate construction, by the blood going to them; for if the blood cease going to them they quickly cease to act in warm-blooded animals. Wundt puts the matter thus: "Slight excitations generally vanish in the central substance of the cell, while stronger excitations set free its latent force. The excitations of the blood-current impinging on the periphery of the cell must accumulate until they reach a given strength sufficient to liberate the force of the cell, when it is thence transmitted to its central region, and thence to its motor nerve. After this discharge of force equilibrium is restored, and the process begins all over again. Nerve centres submitted exclusively to the excitation of the blood, act automatically and rhythmically. This rhythm is interrupted or rendered irregular when the influence of other nerve centres inhibits the discharge of accumulated force." The rhythmic action of the cardiac ganglia, then, is the essential factor in the heart's intermittent movements; force accumulates within the nerve cells of these ganglia, at the time that the sensation of distension of the muscular chambers is received by the afferent fibres, a discharge takes place, which travels along the motor efferent branches and sets up muscular contraction. This simple basal process is modified and controlled (in health), and interfered with (in disease), by the same complex nerve arrangements described above.

By constant play betwixt these nerve arrangements of the heart and the vasomotor nerves of the arterioles, the circulation is kept balanced and adapted to the wants of the organism. Just as the smaller arteries oscillate and vary in their calibre from time to time, so the heart chambers vary. The chambers of the heart are not of invariable and unchanging size, but are sometimes comparatively distended with blood, which condition of distension is readily recovered from, as is well and easily seen in the right ventricle of a lean man, when the breath is momentarily held and recovered again. Disease of the heart may occur from increased demand upon the organ; for a time the tissues grow, the muscular walls and their nervous arrangements growing *pari passu;* but in time the compensating growth comes to a standstill, and then heart failure follows. How this is brought about will be seen in the chapter devoted to the consideration of Hypertrophy and Dilatation. Then again, in consequence of its complex nerve connections, the heart's rhythm may be disturbed by irritation commencing in a far-away organ; thus in women palpitation and irregularity of action may be, and often are, set up by irritation arising in uterus or ovary—a class of reflex cardiac disturbances just commencing to receive the attention they deserve.

When the valves of the heart are injured by disease which renders them functionally imperfect and unequal to completely closing the ostium, then the heart is truly degraded, and lowered to some form less perfect than the four-chambered mammalian heart with its perfect valves. The heart is more or less reduced to the primitive form of a pulsatile sac, and becomes more a mere muscular chamber. The force of the hollow muscle is less perfectly economized by the valves, and so muscular hypertrophy is necessitated—a new balance is struck thereby, which in some cases may be maintained for years, where the valve lesion is small and is static, and therefore the muscular compensation is not great and can be readily maintained. But where the valve lesion is extensive and progressive the case moves rapidly onwards and downwards; the heart ceases to be equal to the demands of the active organism; if the organism could be lowered to the status of a mollusc, and be levelled down to the condition of the muti-

lated heart, life might be maintained without cardiac valves at all; but this is incompatible with the active circulation of a warm-blooded vertebrate. Narrowing, or stenosis of an ostium produces the same muscular changes, a new equilibrium is kept up for a time, but its duration is limited; it can never, under the best circumstances, equal the perfect, healthy, and normal equilibrium.

In order, however, to comprehend more clearly the effects of valve lesions upon the muscular walls of the heart, it is necessary to bear in mind the fact that the valves are not just barely large enough and competent to close the ostium to which they are attached—they are rather more than competent, and can undergo a certain limited amount of contraction or shrivelling before they are rendered insufficient, or the heart may be temporarily overdistended and the ostia enlarged therewith, without the auriculo-ventricular valves leaking. Wilkinson King, in an article published in 1837, gave an account of a series of experiments made to test the efficiency of the mitral and tricuspid valves. He removed the aortic valves, and then filled the left ventricle by water poured down the aorta. He found the mitral valve invariably competent even under heavy pressure; but the tricuspid readily leaked when similarly tested. From this King evolved the theory of what has been termed "the safety valve action of the tricuspid," an hypothesis that has captivated many minds, but is now dying out. King overlooked the fact that there is a certain amount of ventricular contraction which is requisite to the floating back of the auriculo-ventricular curtains before they are closed, and he could not, nor can anybody else, prove that the tricuspid valves are incompetent after so much contraction, with synchronous diminution of the tricuspid ostium, has taken place. Then again, in animals which seek their safety in flight, there is found a muscular band (the moderator band of Reil), extending from the interventricular septum to the outer or yielding wall, and inserted at the point whence arise the musculi papillares of the tendinous cords of the outer valve flap; so as to prevent regurgitation when the right ventricle is distended by the efforts made, and by the demand upon the right heart. Thus an elaborate arrangement exists to add to the efficiency of the tricuspid

valve in animals where there is great demand upon the right heart. Further, in the diving mammals the walls of the right ventricle are not only unusually strong, equalling the left in thickness, so that the heart is truly double (in the dugong, as said before, resembling a double nut in shape); but there are strong tendinous cords which extend from the interventricular septum to the outer wall, to prevent overdistension of the right heart, and leakage of the tricuspid valve during the strain involved in diving. Even the right auricle is strengthened by tendinous trabeculæ. Tricuspid regurgitation does, unfortunately, take place in man when the right heart is overdistended, without necessarily, in all cases, the tricuspid valves being affected; but it is not a matter for congratulation. Such a thing as a safety valve action of the tricuspid is opposed to all we know of the provisions made for the needs of certain animals, as well as to what we know of natural conservative compensatory changes inaugurated and maintained when the heart is the seat of disease in man.

The heart is an organ which, in most persons, has a certain amount of reserve power in it, *i. e.*, it is not merely equal to the demands of the organism when at rest, but admits of great and sustained efforts being made. But this capacity on the part of the heart to undergo the taxation of effort is not equal in all persons. Some persons possess small and feeble hearts, and consequently their capacity to exert themselves is limited; while others again possess large powerful hearts, which enable them to undergo prolonged and severe exertion without any failure thereof. This is a matter of moment in practice. A slight valvular lesion will cripple a feeble heart; considerable injury may be done to a naturally powerful heart, and yet it may remain a fairly efficient heart, permitting of much general activity for years to come.

Then, again, there is more irritability in the tissues of some persons than others. A valve-change in one person will proceed to utter ruin in spite of every effort made to arrest its progress; in another it will remain static for years, admitting of muscular compensation being readily achieved and easily maintained.

There is much more in the diagnosis of diseases of the heart than the mere recognition of what is revealed by physical examination. It is much easier to detect a murmur than to appreciate

its significance. Having determined that it indicates a valvular injury the next point to be ascertained is this, is the valvular disease static or progressive? On this depends the prognosis, and also the line of treatment to be pursued. For the determination of this point many things are to be considered. One important matter is the natural history, the causation and progress of the particular form of disease before us, and at what ostium it is located. Another matter is the recognition of the accompanying muscular changes; how much has preceded or developed side by side with the valvulitis; how much is the consequential result. Then again, as Latham justly puts it, "The treatment of diseases, rightly considered, is in fact a part of their pathology;" and this is specially true of diseases of the heart. For really useful treatment the function of the heart, its strictly mechanical work, its nutrition, its capacity to develop muscular hypertrophy when a valve is injured, or the work to be done is increased, our power to aid these efforts by the administration of appropriate remedies, all must be borne in mind.

A case of heart disease is not a mere diagnostic puzzle, presenting so many physical signs and certain subjective phenomena to be recognized, registered in a notebook, or even recorded in a textbook. From the fragmentary and passing views presented to us by different cases, we ought to be able to construct, with considerable certainty, a comprehensive view of that disease as a whole; of the past history of each case, and even more than that, we should be able to erect a fairly accurate forecast of its future progress and the patient's prospects. In observing this we must recognize the influence which will be exercised by judicious treatment; the consequences of such treatment; and the unfortunate effects of unsuitable treatment. Rarely can we hope to be able to trace the progress of cases from commencement to termination, except in the severe forms of mitral disease set up by rheumatic endocarditis, or the regurgitant form of aortic disease set up by severe toil. It is from the study of numerous fragments of cases, observations of disease at various points of progress, that a whole must be or can be erected. From our acquaintance with the natural history of each form of heart disease, valvular or other, must we form our diagnosis, of which what is told by physical

signs is but the commencement. On an accurate diagnosis rest alike the prognosis and the line of treatment.

A thorough consideration of the work of the heart, its temporary changes of form, its nutrition, its innervation, our means of acting upon it by agents which influence its nervous arrangements, the laws of hypertrophic growth, the reflex effects of disturbance elsewhere acting upon the heart, the separation of these nervous disturbances from the phenomena which are the result of organic disease, all are essential for a proper comprehension of each case which presents itself before us. In some respects our knowledge of heart disease has retrograded. Much that lies outside mere physical signs has been forgotten. The works of Hope and Latham, full of philosophical considerations of the greatest value, have given place to works more devoted to the consideration of diagnosis and the requirements of the examination table, rather than the needs of the bedside. From this condition there is now springing up a reaction. Heart disease is no longer looked at from the hopeless standpoint of the deadhouse and the evidence of organic disease furnished in life by physical signs. We now comprehend that the most hopelessly unalterable conditions once had a stage when they were more or less manageable and amenable to treatment, except ulcerative endocarditis in septic conditions. Our knowledge now is such that we do not stand by with folded hands while disease is running a course over which we can exert an influence, and supinely wait until irremediable injury has been inflicted, and then try what our knowledge of palliative measures will do to retard the patient's progress to the grave. From our acquaintance with disease of the heart, we do not now wait for the unmistakable and brutal answer of a murmur to tell us that morbid changes are being inaugurated in the aortic valves. The murmur tells us in ominous language that the mischief has been done. We now know that it is not the mere endocarditis set up during rheumatic fever that we have to dread, but the interstitial growth of connective-tissue corpuscles in the fibrous structure beneath, lighted up by the inflammatory storm, but continuing for some time afterwards, whose consequences we have to fear. Pathology, read aright, guides us in practice as to what we must do and what we must avoid, and its

study should not plunge us into hopeless despair. When gazing on the ruin disease has worked in a heart, as it lies before us in the deadhouse, we are not to be carried away with the impression that this destruction is beyond the reach of the remedial art; we should rather be incited thereby to consider the commencement of the morbid process, inspired to reflect on the causation of the disease, that period during which we might have interfered beneficially, when we could have arrested a process which, when once thoroughly established, it is beyond our power to influence.

We must not transfer the hopelessness which attaches to the treatment of the close of a long morbid process to the early stages, when treatment may be invaluable. But for this we must learn to recognize the early stages in time; and in order to do this we must study the natural history of each form of heart disease, learn its causation, and be familiar with its associations. We can do little, and that only palliative, when the full-blown, perfectly-developed disease is presented to us in all its completeness; but we may and can do much at the commencement, if we can only recognize that commencement and are on the alert as to its indications. Such should be the outcomes of our pathological study.

Latham says, with as much truth as elegance of language, "Yet it concerns physicians, above all men, that there should not be a barren knowledge, but that it should claim honor of mankind from a sense of the benefit they receive from it. Far be it from me to contend that every piece of pathological knowledge is to be disparaged or rejected, which cannot at once be made subservient to a practical purpose. The knowledge is to be obtained at all events, and kept ready for use, whether the use come soon, or late, or never. Use, however, is the end always to be regarded, as well philosophically as morally. An age of great increase of speculative knowledge in medicine ought surely to be an age distinguished by some great practical benefit."

CHAPTER II.

THE HEART'S POSITION AND THE MODE OF EXAMINING IT—INSPECTION—PALPATION—PERCUSSION—AUSCULTATION—VOCAL RESONANCE.

The position occupied by the heart in the thorax may be rudely described as a triangular space extending from the second right intercostal interspace, under which lies the edge of the right ventricle, to the fifth left intercostal space, almost in a line with the left nipple, where the left apex can be felt. Its position is altered by deep inspiration and expiration, while enlargement gives it a tendency to fall down, especially when the aorta is thickened and atheromatous. Being thus comparatively fixed, we can speak of certain points as the apex beat in the left fifth intercostal space, or the maximum intensity of the aortic second being heard in the second right intercostal space; while a tricuspid murmur is heard at, and usually only at, the ensiform cartilage; while hæmic murmurs at the pulmonary orifice are heard in the third left intercostal space running up to the clavicle. Occupying such a determinate position, the heart admits of being examined by inspection, palpation, percussion, and auscultation.

The auricles are on a line with the third costal cartilages. At the left of the sternum, on a level with the third intercostal space, lies the mitral valve; in front of this, more directly under the sternum, and but a few lines lower down, lies the tricuspid valve. The pulmonary orifice is seated opposite the junction of the cartilage of the third rib with the left edge of the sternum. Near it, very slightly lower, but placed more obliquely, are the aortic valves. (Da Costa.)

Inspection tells us how far the patient's physique is good; whether the intercostal spaces are widened or narrowed by inspiration; whether each side of the chest is playing well, and if the movements of the chest are symmetrical. For this purpose a good light is required, and the patient should face the light, a position which throws the medical man's face into the shade; a

matter often of no slight importance. When the light thus falls on the thorax a faint regular pulsation may be seen at the fifth intercostal space. In some persons with solid ribs scarcely any movement may be observable in health; while in thin persons under excitement this rhythmic movement may extend over a space the size of the palm, without there being any disease present.

The cardiac impulse is modified by certain abnormal conditions. It is increased by hypertrophy, and still more by hypertrophy and dilatation, especially in young persons. In left-side hypertrophy the apex beat is carried downwards and outwards to the left, even beyond the line of the nipple; in right-side hypertrophy it is felt more towards the ensiform cartilage. In simple dilatation the impulse is less heaving and more diffused. It is diminished in fatty degeneration, in cases of emphysema with rigid costal cartilages, and in pericardial effusion. In chorea, in Basedow's or Graves's disease, and in simple palpitation the cardiac impulse is greatly magnified, and the appearance of hypertrophy simulated. The apex beat may be elevated by abdominal effusion or pericardial effusion (Walshe); an enlarged left lobe of the liver; or it may be dragged up by retraction of the lung after the falling in of a cavity; it may be depressed by enlargement of the heart, aneurism, or a tumor. It may be pushed over to the right by a pleuritic effusion, or a left-side empyema; or it may exist on the right side congenitally. It may be pushed to the left by right-side pleurisy, enlargement of the lung, emphysema, cancer, etc., or pericardial effusion, which always carries the apex beat to the left. (F. Roberts.) Hypertrophy gives the apex beat length, and dilatation width; and in simple dilatation, the impulse is often wide and diffused, but wanting in force. In some cases of right-side dilatation a distinct pulsation may be seen in the right second intercostal space indicating enlargement of the right auricle; while enlargement of the left auricle in mitral disease will sometimes produce pulsation in the left second or third intercostal spaces. (Sansom.) The intercostal spaces will be seen to bulge in pericardial effusion; or there may be a distinct systolic retraction in pericardial adhesion. In pericardial effusion affecting the phrenic nerves and paralyzing the diaphragm, there

is retraction on inspiration, and protrusion on expiration, the diaphragm no longer taking part in the respiratory movements, but merely being affected by them. In some cases the veins of the pericardial region are enlarged.

Pulsation of the liver may be present in tricuspid regurgitation. Then the hue of the skin may teach us something: as pallor, indicating arterial anæmia; blueness, venous fulness or cyanosis; while a yellow tinge may be found indicating venous congestion of the liver, after the tricuspid valve has failed.

Palpation is the application of the hand to the cardiac area, and often furnishes valuable information in addition to what is afforded by inspection. "Many an impulse can be felt that cannot be seen." The beat may be increased by excitement, as well as by enlargement of the heart. In conditions of debility, in fatty heart; in cases of rigid ribs, with a piece of lung, emphysematous, or other, betwixt the heart and the chest-walls; and in pericardial effusion, the heart's impulse is diminished. When the pericardium adheres to the heart the systolic beat may be split up into several parts. "The sounds of the heart can be analyzed by means of the touch. They will be felt, the one as a long and dull, the other as a short and distinct vibration." (Da Costa.) A thrill may be produced either by a roughened pericardium with a to-and-fro motion; or it may be distinct and presystolic as in mitral obstruction. At other times it may be diastolic and felt at the base, when it indicates aortic regurgitation. Or it may be found more rarely with aortic obstruction; and is often present in aneurism of the ascending aorta. Besides the information thus furnished, palpation may be utilized to educate the senses as to the recognition of the changes produced by right-side dilatation. If a finger be placed on the apex beat of a thin muscular person, and he be told to hold his breath, the apex beat will be gradually lost, as the right ventricle becomes distended with blood; when the person once commences to breathe again, "the water-cushion" (W. T. Gairdner) can be felt passing away until the apex beat is once more distinctly felt. Such a condition is produced, of a less evanescent character, in some states of right-side engorgement. Palpation will tell much about the modifications of rhythm of the heart, a subject to be considered further on. Pal-

pation will also tell of the "jogging motion" of Hope, or of the embarrassed heart where the pericardial adhesion involves the costal pleura. In a simply dilated heart there is something pathognomonic in the few rapid and feeble strokes preceding a halt; and then the sensation of "rolling over" as the systole proceeds with a more than ordinarily powerful contraction.

There is something indescribable by words which distinguishes the feeble, quiet, undemonstrative action of a fatty or atrophied heart from the obvious struggling action of a heart structurally sound but dilated. Skoda says, aneurism of the left apex may indicate itself by fulness and protrusion of the intercostal space over the left apex at each systole, from the aneurismal portion being pushed forward.

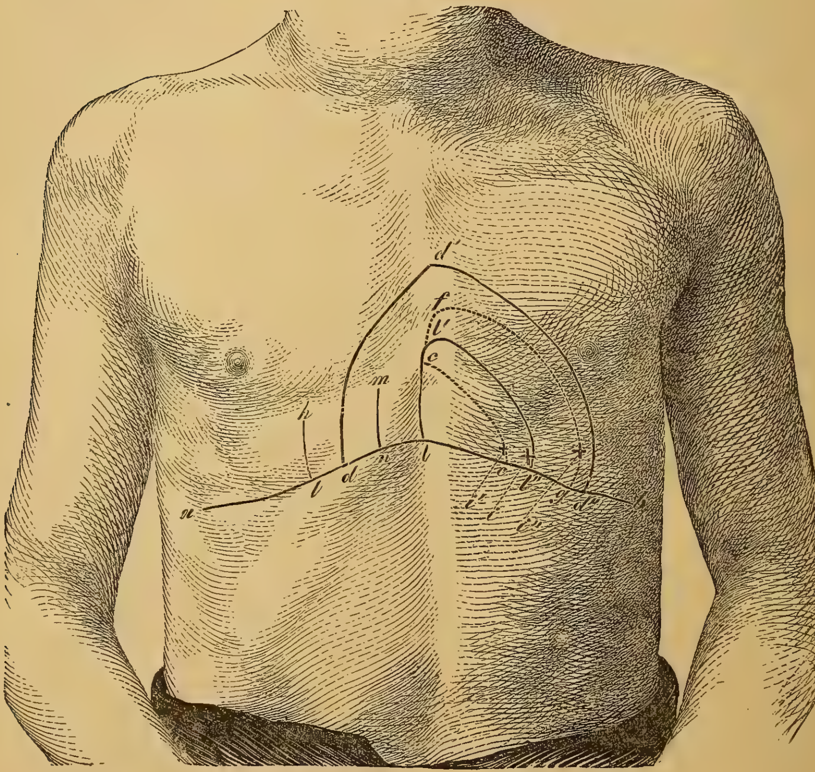
Percussion is the means by which we estimate the extent of cardiac dulness. The area of complete dulness is a triangular space where the right ventricle is uncovered by lung. Around this is a border of gradually lessening dulness, as the amount of lung betwixt the heart and the chest-wall deepens. To ascertain this area it is well to commence near the middle of the left clavicle, and percuss steadily downwards; at the lower border of the fourth costal cartilage, a distinctly dull sound, accompanied by a sense of decided resistance, tells that a solid organ is reached. On the right side, the border of the heart dulness is found at the fifth rib near the sternum, the right lung covering the right auricle and much of the ventricle. The apex is reached by advancing in an oblique direction from the right border. But we can save ourselves that trouble by feeling for the impulse, or listening for it with a stethoscope. The inferior surface is to be ascertained by prolonging the line of dulness on percussion of the upper border of the liver, and then judging by the greater amount of resistance and the fall in pitch, that the heart has been reached. If the other boundaries have been correctly drawn the size of the heart can be accurately estimated—accurately enough, at least, for any practical purpose. (Da Costa.) Walshe, whose accuracy of diagnosis is universally admitted, says, "The heart's superficial area must be as follows: On the right a vertical line, extending at mid-sternum from the level of the fourth rib to that of the sixth; on the left an oblique line passing outwards and downwards at a

more or less acute angle from the latter opposite the fourth cartilage, and curving inwards again (somewhat within the site of the nipple) to the sixth rib, beside the heart's apex; inferiorly, a line gently sloping to the left, from the central point of the lower edge of the sternum, along the sixth cartilage. This is the extent of heart uncovered by lung in calm respiration, and the form of the part is obviously, though only rudely, triangular. The vertical side of the triangle equals about two inches in length in persons of middle stature, the horizontal about two inches and a half, the oblique about three inches." However accurate we may strive to be, it is obvious that the size of a heart cannot be determined absolutely by the area of complete cardiac dulness. If this last area be confessedly difficult to map out, it is still more difficult to ascertain with any attempt at precision the outline of the heart buried under lung—which may be of a greater or less resonance, as in emphysema on the one hand, and consolidation on the other. The area of complete dulness is altered by position, as the accompanying plate from Professor von Dusch's work shows.

The best thing each student can do is to take a series of healthy persons, to fix the exact spot of the apex beat, then to draw on the skin the boundary of a normal heart, making the right auricle touch the right second costal cartilage, which will give the correct position and size of a healthy heart. Having done this he should then industriously percuss the whole region, and familiarize himself not only with the percussion-note of the different organs, heart, liver, and lung, but with the sense of resistance, which is so valuable an auxiliary. Such practice will soon teach him to estimate the area of comparative dulness outside and around the area of complete dulness. In women percussion is of less value than in men in consequence of the mammæ. In children, too, there are difficulties. Practice and patience alone will endow a student with the power to estimate the outline of the heart fairly correctly by percussion; but the power is well worth the trouble required to attain it. It is practically possible in the great majority of cases to estimate with fair accuracy the size of a heart; but where there are rigid costal cartilages, with emphysema of the anterior flaps of the lungs, as seen

in some elderly persons, there is no area of dulness, complete or comparative, that can be made out at all. Nothing but individual experience can overcome the difficulties presented by this last class of cases.

FIG. 14.



a b, Line of diaphragm; *d d′ d″*, Outline of heart dulness when lying on the back; *h l*, When on the right side; *m n*, When on left side; *l l′ l″*, Limit of complete dulness when on the back; *l f g*, When on the left side; *l c e*, When on the right side; *i*, Point of apex beat when on the back; *i′*, On right side; *i″*, On left side.

Not only does position, as seen above, alter this area of cardiac dulness, but it varies with the respiration; is less on full-drawn inspiration and increased by complete expiration. In some persons the amount of left lung covering the heart is less than normal, and here the area of complete dulness is enlarged without

any change in the heart itself. Then there is a hæmic murmur in the pulmonary artery from its being in contact with the chest-walls without a piece of lung interposed. Then the position may be altered, as when the heart is on the right congenitally, or is pushed over by left pleuritic effusion, or too far on the left from right pleuritic effusion, or possibly pushed up by enlargement of the liver. It may be increased in size by enlargement of the heart, either hypertrophy or dilatation ; by pericardial effusion or a mediastinal tumor, or by an accumulation of fat around or upon the heart. Less frequent causes of apparent increase in the area of cardiac dulness are aneurism of the ascending aorta, sub-sternal abscess, or a thymus gland still remaining. Conditions of the lung affect it. Thus consolidation of the lung by pneumonia or tuberculosis will increase it; or deficiency of lung, or retraction of lung over it, will add to it. Fat or thick muscles, and, in the female, the mammæ increase it, apparently that is. It is diminished by atrophy of the heart, the lung encroaching on the space undoubtedly; it is lost in pneumopericarditis, indeed, this area then becomes tympanitic ; it is lessened when the lung is bound down over the heart by a pleuritic adhesion, and by an emphysematous condition of the anterior portion of the lungs. The shape of it varies too; in hypertrophy it extends downwards beyond the normal line; in dilatation it is more extensive generally, and is square or circular, while in pericardial effusion it is triangular, with the base down, or, according to Sibson, it is pear-shaped, with the broadest part lowest and the heart and vessels at the narrow end above. It changes with posture. In right-side enlargement the lung may be pushed away until the area of dulness extends to the third rib, and the right auricle can be felt pulsating. The area of cardiac dulness is greatly modified in the rare cases of diaphragmatic hernia. Changes in the size of the cardiac area may be utilized as tests of the freedom of play of the edges of the lungs. (Walshe.)

Auscultation is the most perfect means we possess for ascertaining the condition of the heart, either in health or in disease. We can ascertain thereby at once the state of the heart-walls, whether normal, hypertrophied, dilated, or the seat of fatty degeneration; we can ascertain if the rhythm be perfect or not; we

can hear the normal sounds of the valves, or if disease be present recognize obstruction or regurgitation, while the impulse transmitted by the stethoscope is often of great service. All this can be done, provided the auscultator be equal to it. The more valuable this means of examination the greater the pains required to become perfectly familiar, first with the normal sounds, and then with the abnormal sounds. The normal sounds of the heart are two: the first sound, corresponding in time to the systole or contraction, is comparatively long; the second, occurring during the diastole, is short and sharp. Betwixt the first and the second sound there is a brief interval or pause; betwixt the second and the succeeding first sound there is a comparatively long pause, during which the cardiac chambers are being filled with blood. The right ventricle is filled by blood coming in from the venæ cavæ; the left by blood coming in from the pulmonary veins. The blood flows into the empty chambers until the internal pressure within the cardiac chambers and the veins behind is equal. "The ventricle (right) thus being filled the play of the tricuspid valves comes into action, the auricular systole is followed by that of the ventricle, and the pressure within the ventricle, cut off from the auricle by the tricuspid valves, is brought to bear entirely on the conus arteriosus and the pulmonary semilunar valves. As soon as by the rapidly increasing force of the ventricular contraction the pressure within the ventricle becomes greater than that in the pulmonary artery, the semilunar valves open, and the still increasing systole discharges the contents of the ventricle into that vessel. But as the systole passes off, the pressure in the artery becomes greater than that in the cavity of the ventricle, and a rebound of blood takes place. The first act of this rebound, however, is firmly to close the semilunar valves, and thus to shut off the overdistended artery from the now empty or nearly empty ventricle. During the whole of this time the left side has with still greater energy been executing the same manœuvre. At the same time the venæ cavæ are filling the right auricle, the pulmonary veins are filling the left auricle. At the same time that the right auricle is contracting, the left auricle is contracting too. The systole of the left ventricle is synchronous with that of the right ventricle, but executed with greater force, and the flow of

blood is guided on the left side by the mitral and aortic valves, in the same way that it is on the right side by the tricuspid valves and those of the pulmonary artery. The beat of the heart is a comparatively slow long-continued spasm, and not a tetanic convulsion." (M. Foster.) In the frog, the systole, commencing in the auricle and running into the ventricle, forming a truly peristaltic action of a rapid character, is well seen. A certain amount of time is requisite for each cardiac cycle, which is divided into the systole, or period of contraction, and the diastole, or time when the chambers are being filled. This time has been variously estimated.

According to Hermann, "The systole of the ventricles takes up about two-fifths, their diastole about three-fifths of a complete cardiac revolution. (Valentine, Landois.) This, however, is only when the pulse is of normal frequency, since any change in its rate only varies the duration of the diastole, while that of the systole remains constant. (Donders.)" Instead of giving any engraving of the cardiac cycle, it is sufficient to say that the cardiac revolution comprises a first sound, a brief pause, a short second sound, and a long pause. If the pauses be represented by dotted lines and the sounds by unbroken lines, a cardiac cycle would stand so . . . . . ——— . . —, the first long pause representing the filling of the chambers; the long line the systole; the second pause the time of arterial distension; and the short line the closure of the semilunar valves (pulmonary and aortic), by which the reflux of blood into the ventricles is arrested. This consideration will enable us to readily comprehend the causes of the sounds of the heart. The first sound is comparatively long. It is generally admitted to have two causal factors, viz., the muscular contraction and the closure of the auriculo-ventricular valves. To these it has been proposed to add the impact of the blood in the contracting ventricles upon the blood already in the aorta and pulmonary artery. (Leared.) In some cases the muscular sound preponderates; in others the slap of the closure of the mitral and tricuspid valves is very distinct. In order to get a good conception of the muscular sound the student should apply his stethoscope over the heart, and then upon the ball of his thumb, putting the muscles of the thumb into rhythmic action.

This can be done without any movement of the skin, which might occasion a friction-sound as a source of error. Having so familiarized himself with the character of the muscular sound, the young auscultatôr will soon acquire the power to discriminate the muscular sound from the other factor, the "slapping" sound of the closure of the auriculo-ventricular valves. In hypertrophy the second is dull and muffled; while in dilatation the thin wall emits but sharp short muscular sounds, and the flapping of the auriculo-ventricular valves is distinct. In hypertrophy with dilatation these valves are sometimes driven together so violently as to produce a ringing sound. It is clear that the first sound may be modified by changes in the muscular walls, or by changes in the valves. If the edges of the valves be thickened or contorted by a slow inflammatory process, the sound produced will be modified, even when the change has not progressed so far as to produce a murmur, either obstructive or regurgitant. This first sound is increased by excitement, and is diminished after illness, in conditions of debility and by fatty degeneration of the heart-walls. It is increased by consolidation of the lung over it, or by retraction of the lung so that an abnormally large portion of the lung comes in contact with the chest-walls; it is lessened by an emphysematous state of the lung over the heart, until sometimes it can scarcely be heard. It is diminished by pericardial effusion or collection of solid material in the pericardial sac. (F. Roberts.) As to the pitch or quality of the first sound, it is dull or obscured by hypertrophy, while it is short and clicking in dilatation. In anæmic states both the first and second sound may be clear and high-pitched. At times the first sound is heard much more distinctly at the apex than the base, not only the valve-sound but also the muscular sound. What modifications are produced by the impact of the blood in the cardiac chambers upon the blood in the arteries, if any at all, and if so, of what nature, cannot yet be stated. The blow of the heart upon its parietes by the elongation of the arteries, or distension by the ventricular systole, has been stated to effect the first sound; while the Germans attach importance to the rushing of the blood through the aortic and pulmonary orifices as a factor in its production; but we know nothing of these as causes of modifications of the sounds of the

heart, except in the production of murmurs, though the former undoubtedly affects the impulse.

Some statements of Dr. Frederick Roberts as to auscultation of the heart are so important that they are worthy of verbatim quotation: "At the left apex, *i.e.*, just within and below the nipple, the systolic sound is prolonged and well defined; much accentuated; it seems muffled and rather deep, and of rather low pitch. The diastolic is much shorter, sharper, and more abrupt; clearer; more superficial, and higher pitched. Comparing the sounds at the base and apex, it will be found that at the base the diastolic sound becomes more marked. It is loud and distinct, harsh and accentuated, clear and often ringing; while the systolic sound is dull and indefinite, shorter, and without any accent. At the right base, *i.e.*, opposite the second space or third cartilage, close to the sternum, the sounds are usually louder than at the corresponding point on the left side, especially the diastolic. Finally, it must be noticed that the sounds are generally better heard under the left clavicle and over the left side posteriorly than over the corresponding regions on the right side. It is often of advantage to compare the sounds over different parts of the thorax, but especially at the apex and the base of the heart, and at the right and left apex or base. As illustrations of the knowledge thus to be gained the following are important: 1. If the sounds, being weak at the apex, are louder at the base, this serves to distinguish pericardial effusion from dilatation or fatty heart. 2. Greater intensity at the right apex than the left either shows displacement of the heart, or that it is covered by some imperfectly conducting material, especially an emphysematous lung. 3. Louder sounds at the left base than the right, especially the diastolic, indicate that there is some disease affecting the passage of blood through the mitral orifice, so that the pulmonary circulation is overloaded and the pulmonary artery distended. 4. Any condition, either in connection with the heart itself or external to it, which alters the position of the organ, will correspondingly modify the sounds. For example, in left pleuritic effusion they are transferred to the right side of the chest. 5. The extent and direction of conduction of the sounds may be useful in determining disease in other organs. Thus in consolidation, at the apex

of the right lung they are very often louder under the right clavicle than the left. In right basic pneumonia they are frequently very marked over the affected part. Cavities in the lungs may intensify the sounds considerably, or sometimes impart to them unusual characters, such as hollowness or a metallic quality. In auscultating the heart in order to detect abnormal conditions, it may be necessary to make the patient stop breathing for a moment, and it is often advisable to excite the heart by a little brisk movement. In order to compare the sounds at the base and apex some authorities recommend the use of a double stethoscope, so that they may be heard simultaneously, but the ordinary instrument answers perfectly well."

The second sound of the heart is now attributed entirely to the closure of the semilunar valves.* The aortic second sound is stronger and thicker than that produced by the pulmonary valves. The aortic second sound is heard most clearly at the second right costal cartilage, close to the sternum, where the aorta comes nearest to the anterior chest-wall; but it may often be heard with great clearness even at the apex of the heart. This sound is produced by the aortic recoil, and is thus proportioned to the blood-pressure in the arteries. Consequently it is commonly loud and accentuated in conditions of chronic Bright's disease, where it is of great diagnostic value. (Rosenstein.) This, of course, is more the case where the vascular system is chiefly implicated than in those cases where the circulatory organs are little affected. It may be merely loud or hard, like the contact of two metallic bodies, or it may be clanging. Accentuation of the aortic second sound may be "booming" even in some cases of aortic aneurism. (Warburton Begbie.) It has also been observed in the general paralysis of the insane. Here I have found it most marked in the earlier stages of the complaint, when the general nutrition is good, and the heart shares therein. It is sufficiently marked often to carry with it a diagnostic value. In some cases of anæmia the aortic second sound is very clear and distinct. The pulmonary second sound is heard most clearly at the left third costo-sternal

* Sibson ascribed it to tension of the whole root of the aorta, not merely to the tension of the aortic valves, the base of the chamber.

articulation, and is usually heard only over a very limited area. It can usually be readily distinguished from the louder aortic second sound. The pulmonary second sound is accentuated when the pulmonary circulation is obstructed, as in chronic bronchitis with emphysema, fibroid phthisis, and in mitral disease; indeed, under those circumstances where the right ventricle becomes enlarged. The loudness of the pulmonary second sound is held by Skoda to be a better and more trustworthy index of the extent of a mitral regurgitant lesion than the loudness of the accompanying mitral murmur. Cases have occurred to the writer where an accentuation of the pulmonary second sound was heard along with pulmonic congestion, and which soon afterwards developed acute pulmonory tuberculosis. The aortic second sound is modified by a thickened edge of the aortic valves; and such muffling is not rarely heard before the valvulitis has led to the production of a murmur.

Reduplication of the sounds of the heart, both first and second, are not rare, but the diagnostic value of the phenomenon has not yet been determined. It indicates that the ventricles do not act synchronously. George Johnson holds that, in reduplication of the first sound, the sounds of the auricular systole are heard, as well as those of the ventricular systole. While Hayden states that this reduplication "is due to resolution of that sound into its two normal elements, namely, the cardiac impulse and the sudden tension of the auriculo-ventricular valves."

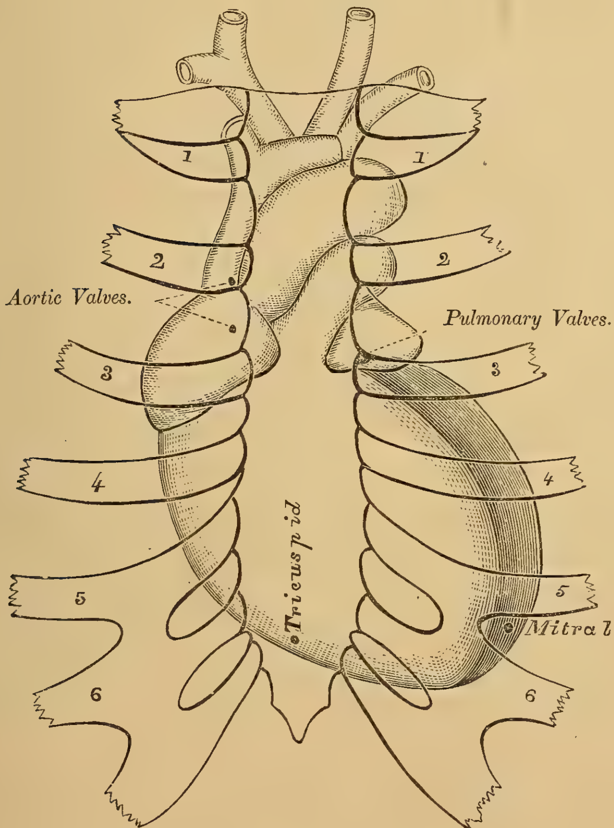
Murmurs.—Of all the physical signs of disease of the heart the most significant are murmurs. The blood does not normally produce any sound in rushing through the different ostia of the heart. Under certain abnormal circumstances it does produce sounds, which sounds are of great diagnostic value. These are alterations in the ostia, or the valvular flaps connected therewith. They may consist of narrowing of the ostia with roughness of the valvular curtains, producing obstructive murmurs. Or the vela or cusps may be distorted or mutilated by inflammatory action, and so permit of regurgitation of the blood; or an aortic cusp may be torn down by violent effort. Such regurgitation usually produces a soft murmur. Obstructive murmurs are usually harsher than regurgitant murmurs, though some aortic regurgi-

tant and mitral regurgitant murmurs are loud and distinct. The obstructive murmur is commonly like the sound of a saw going through a piece of soft wood. The regurgitant murmur has usually a blowing sound. The various names of murmurs in which the French school of a generation ago delighted are not now much used.* A musical murmur is sometimes heard, and is said to be due to a shred of lymph hanging from the free edge of a valve and vibrating in the blood-current. It is stated that no cardiac murmur is ever heard by the patient. In a case of a very loud murmur, which could be heard three inches from the patient's chest, he could hear it himself, and on listening with a binaural stethoscope recognized the murmur, only louder. It was a case of rupture of an aortic cusp from effort, and dated back eight weeks. To take the murmurs of organic change first, we can see that each valve may have two murmurs associated with it, viz., an obstructive and a regurgitant one. In distinguishing them their time and their site of maximum intensity are of great value. The great divisions as to time are the systolic, the diastolic, and the presystolic or prediastolic. As to area of maximum intensity, as a crown piece would cover the four valvular orifices of the heart, it is obvious that this would not be of much service if it were not that each murmur travels a little in the direction of the blood-current which evokes it. Thus an aortic obstructive is conveyed along the aorta forwards; an aortic regurgitant backwards towards the base of the heart, down the sternum; while a mitral obstructive is loud at the right apex, and a mitral regurgitant is carried backwards, behind the left nipple, on to the axilla. A pulmonary murmur is transmitted towards the left clavicle. Tricuspid murmurs are faint, and only heard over a limited area at the xiphoid cartilage. The accompanying plate illustrates the points where the murmurs of the different valves are most distinctly heard, and marks the various areas. It is taken from Da Costa's well-known work on "Medical Diagnosis," with a slight modification as to the position of the pulmonary valve sound.

* "Upon the whole, my persuasion is, that no practical good has come from curiously naming and noting, and multiplying endocardiac murmurs."—P. M. Latham.

Da Costa places it over the second left costo-sternal articulation, while the writer holds with W. T. Gairdner in placing it over the third left costo-sternal articulation.

FIG. 15.



(From Da Costa.)

The systolic murmurs are four. They include obstructive murmurs of the pulmonary and aortic orifices, and regurgitant murmurs of the auriculo-ventricular valves. They may be arranged so as to make them clearly distinct as follows:

VALVE.	MURMUR.	TIME.	CHARACTER.	POINT OF MAXIMUM INTENSITY.
Aortic.	Obstructive.	Systolic.	Rasping.	Second right costo-sternal articulation.
Pulmonary.	"	"	"	Third left costo-sternal articulation.
Mitral.	Regurgitant.	"	Blowing.	Left apex, to left.
Tricuspid.	"	"	"	Limited to ensiform cartilage.

These murmurs are all heard with the first sound, and alter it or obliterate according to the character of the murmur. Sometimes the sound of an aortic obstructive is so loud as to be clearly heard over the whole cardiac area. They are simulated by hæmic or dynamic murmurs, heard chiefly at the pulmonary and mitral orifices. Pulmonary hæmic murmurs are common in conditions of anæmia, and are found along with the *bruit de diable.* They are heard at the pulmonary orifice and upwards to the left clavicle, inclining slightly outwards to the shoulder. Organic pulmonary systolic murmurs are so rare that a pulmonary murmur is all but invariably hæmic, except when it arises from a congenital defect or fœtal change. Its accompaniment also indicates its nature. Then a pulmonary systolic murmur may be found under other circumstances, viz., where the lung does not sufficiently cover the heart. Here the murmur may be very loud. Its great characteristic is the influence exercised over it by inspiration. A long-drawn inspiration, by inflating the lung and thus interposing more of it betwixt the heart and the chest-wall, lessens the murmur markedly, while full expiration, bringing more of the heart and pulmonary artery in contact with the chest-wall, intensifies the murmur. Such a murmur may be found along with evidences of cardiac debility, and when so found is a great stumbling-block in diagnosis.

Non-organic mitral regurgitant murmurs have been recognized by writers since Laennec's time, who said, "I can state with certainty that the bellows' sound of the heart is very often met with when the organ is perfectly healthy." The explanation offered of such murmurs is that they are due to irregular action of the musculi papillares preventing the complete closure of the mitral vela, and so permitting of a regurgitant murmur; while Da Costa

thinks they may be due to tension of the mitral valve in conditions of excitement of the heart. George Balfour, in speaking of curable mitral regurgitation, says of a murmur, first accurately described by Naunyn, that it is auricular in its seat. It is found "about one inch and a half, or rather more, to the left of the pulmonary area, and in the same place immediately over the part where the appendix of the left auricle pops up from behind, just to the left of the pulmonary artery. This so-called arterial murmur is, therefore, not arterial at all, but strictly auricular in its source." Temporary mitral murmurs are heard in chorea, in Graves's disease, after violent effort, and more rarely under other circumstances.

Of organic mitral murmurs, systolic in time and regurgitant in character, it is found that in some cases the murmur does not travel in the direction of the nipple, and away to the left of it, so that it may even be heard at the median line at the back, but rather takes the direction of the axilla. It has been stated that when a mitral regurgitant murmur takes the direction of the axilla, the aortic flap of the valve is chiefly affected, while if the murmur be heard away to the left of the nipple, and even further backwards, it is the outer mitral flap which is chiefly involved. Usually a mitral regurgitant murmur is heard in a direct line from the left apex to the left, and backwards to the spine even when loud.

The tricuspid regurgitant murmur is heard only over a very limited area, lying betwixt the right apex and the ensiform cartilage. It is often wanting where regurgitation is present.

During the time of ventricular inactivity are heard the diastolic murmurs, and the postdiastolic, or, as it is now usually termed, the presystolic, corresponding to the time of the auricular systole. The diastolic murmur is that of aortic regurgitation, for pulmonary regurgitation is practically unknown. It is heard down the sternum, and is usually loudest at the fourth interspace. It may, however, be louder somewhat lower down, and varies as to locality according as it is pronounced or faint, and, as will be seen in an after chapter, according to which aortic cusp is most affected. It is blowing in quality, and varies considerably in duration in different cases. It is very commonly found along with

an aortic systolic murmur, and in such cases the murmurs will sometimes vary from time to time; at one examination the obstructive sound is the chief one heard, while at another time the regurgitant murmur preponderates. Usually an aortic regurgitant murmur is heard only over a limited area, and very seldom at the back; at times, however, the double aortic "to-and-fro" sound is heard so distinctly at the mitral area that it suggests, at first, the impression of a "button-hole" mitral, especially when the mitral area is auscultated first.

The presystolic murmur is practically a mitral murmur. Tricuspid stenosis is very rare, and when it produces a murmur it is presystolic in time, and heard at the fifth intercostal space, an inch to the left of the sternum. (Hayden.) The presystolic murmur of mitral stenosis has recently excited much attention in Great Britain. It is heard at the heart's apex, and is sometimes conveyed a little to the right of the apex. It is not heard away to the left or to the axilla, as is the mitral regurgitant murmur. Dr. Hilton Fagge, who has paid great attention to this murmur, says it has a "churning" or "grinding" character; that it may be very short in some cases; that the first sound of the heart is often peculiarly sharp and clear, so as to be taken for the second sound. "This murmur is much louder at the heart's apex than anywhere else. It is also remarkably local, being sometimes audible only at a single spot, and not being traceable round the side of the chest towards the left scapula, as is the case with the systolic murmur of mitral regurgitation." Some further points connected with this murmur will be considered when mitral stenosis is discussed.

Beyond these well-recognized murmurs are heard others, associated with congenital malformations. They are usually basic; that is all that may be affirmed about them, and they are chiefly recognized by their negative characters, not corresponding to any of the well-known murmurs.

Murmurs have been heard where no morbid change could be found after death. At other times pronounced lesions are found where no murmur could be detected in life. This is most liable to occur where a tricuspid lesion exists, and the blood-current passing through the altered mitral or aortic orifices is too small to

bring out a murmur. Murmurs will intermit and disappear for intervals in a very perplexing manner. I once heard a loud systolic basic murmur in an epileptic at the time of the attack, which could not be detected after the fit was over. Even the aortic regurgitant, the most persistent of all, has been known to be absent for distinct periods. (W. T. Gairdner.) Peacock states that anæmia will sometimes magnify an organic murmur in a very perplexing manner. Treatment, by improving a dilated heart, will sometimes bring out a mitral murmur which could not be heard previously. On the other hand, mitral regurgitant murmurs have been known to disappear as the dilated chamber and its ostium become restored to their normal size; and so the valvular curtains, not extensively diseased, are once more equal to the closure of the ostium.

Mensuration of the thorax will give no information that has not been more amply furnished by other means.

In addition to the ordinary means of examining the heart, Flint adds vocal resonance. He says, "The boundaries of the heart may often be as accurately defined by auscultating the voice as by percussion, and in conjunction with the latter method the former may be resorted to in determining the augmented space which the heart occupies in cases of enlargement. In females often, owing to the size of the mammæ, the diminution or extinction of the vocal resonance is more available in determining the area of the superficial region than dulness on percussion." There is a common sense in this which at once recommends it to the reader.

There are some other points to be attended to in examining the condition of the circulation which will next be considered, and the information furnished thereby is of scarcely less importance than the matters just discussed.

CHAPTER III.

OBJECTIVE SYMPTOMS—PALPITATION—IRREGULARITY—INTERMITTENCY—THEIR DIAGNOSTIC VALUE—AID DERIVED FROM THE ARTERIAL AND VENOUS SYSTEMS—EFFECTS OF EFFORT—THE SPHYGMOGRAPH.

In perfect health the heart carries on its work without producing any consciousness of its existence, except after violent effort. It has so much "spare power" about it, like many other organs, that it can meet the ordinary demands upon it and something more, without any evidence of tax upon it. Severe exertion, however, causes it to beat so violently that its action is felt distinctly. Such excited action is more readily produced in some persons than others. Some seem to have so much "spare power" in their hearts that long-sustained effort even does not produce much cardiac disturbance. In many of these persons there is a large heart, beating slowly, which can stand without strain almost any amount of effort; yet such a condition has been mistaken for disease and the heart been pronounced to be hypertrophied, and the owner of it directed to be careful. A like condition is found in animals. "Eclipse," as a racehorse, and "Master McGrath," as a greyhound, were each remarkable for their great endurance even more than their speed; in both after death the heart was found to be of unusually large size. On the other hand, some hearts seem to possess little spare power, and are readily excited by slight provoking causes, such as do not affect the hearts of ordinary persons. But we do not regard these objective symptoms of the heart's existence and its functional labors as the necessary evidence of disease. Palpitation may indicate disorder or it may point distinctly to disease. Of the two it is more common in disorder; in disease it is most commonly induced by effort. As severe effort brings out palpitation in healthy hearts, so slight effort will elicit palpitation, as an evidence of adynamy, in diseased, and especially dilated, hearts. But there are different forms of palpitation, that is, it is due to different causes, and its significance varies with its causal associations.

In bypast days, when hypertrophy of the heart was held to be a spontaneous overgrowth till the heart became dangerous from its strength, then palpitation was regarded as overaction. There is a grain of truth in each statement, but it is buried under a load of misconsceptions. When the hypertrophy is sufficiently developed as a compensatory growth, palpitation is comparatively rare. When hypertrophy, though present, is unequal to a new equilibrium of fair perfection being set up, then palpitation is common. Here palpitation is readily induced by effort; if the old view were correct, this is just the time when the overaction of hypertrophy should be neutralized and met by demand upon the heart. Hypertrophy is not invariably an unalloyed good, as we shall see; and palpitation is not always the active evidence of taxation. But, as a rule, hypertrophy is the means by which a new equilibrium is attained when the normal pre-existing one is disturbed; any palpitation in organic disease of the heart is the evidence of muscular inefficiency.

Palpitation is less serious in its significance than irregularity of the heart's action; and intermittency is more serious than either. Be it understood that these objective phenomena are here spoken of in connection with disease of the heart; all are at times mere nervous phenomena of little or no significance. Palpitation may manifest itself under three different sets of circumstances; its indications vary with its causal relationships, and so, as we shall see, does its treatment. It is found (1) where the muscular power of the heart is unequal to the demands upon it. It is thus found along with dilatation of the heart, and is here produced by the slightest effort in marked cases. The moment there is any demand upon the heart palpitation is set up, and excited stroke takes the place of the quiet stroke of the normal heart. The dilated heart may beat steadily while the demand upon it is near the minimum; let the demand be increased and then the active evidence of debility is furnished. It is also common (2) where the blood pressure in the arteries is suddenly raised, and thus there is more obstruction to be overcome on each ventricular systole. Consequently it is found in attacks of hysteria. Here we have violent palpitation, a small cordy pulse, cold extremities, and on the cessation of the attack a copious flow of urine of pale

color and low specific gravity—the bulk of urine being the measure of the high blood pressure in the arteries during the attack; during the interval there are evidences of great mobility in the vasomotor nerves, and in the attack there is a spasm of the muscular walls of the arterioles, with great arterial tension and consequent palpitation. It is also found in gouty conditions and along with chronic Bright's disease. (The intimate relations of cardiac conditions to the state known as lithiasis, or as Murchison calls it, lithæmia, will be demonstrated as this work progresses.) The modifications in the muscular walls of the arterioles in chronic Bright's disease, has been demonstrated by George Johnson, Grainger Stewart, and others; while the great rise in the blood pressure in the arteries in this condition was first shown by Traube. Arteriole spasm, excited by imperfectly depurated blood, leads to hypertrophy of the muscular walls of the arterioles, and this change favors further spasm. The subjects of chronic renal changes are liable to vasomotor disturbances, as flushings, and sudden icy-coldness of the hands. When arterial spasm suddenly raises the blood pressure in the arteries, and the left ventricle has to meet a rapid increase in the obstruction offered by the blood already in the arteries, then palpitation is set up; and that too at times even when the ventricle is hypertrophied. Palpitation so associated is, however, not nearly so common in men, where the hypertrophy is usually free from any dilatation of the chambers of the heart, as in women, where the hypertrophy is usually blended with dilatation. In women of middle age or older, palpitation is generally so associated, and arises at other times than on, or after, effort. A slight contraction of the arterioles will excite palpitation when the patient is sitting quietly, or may even wake her out of sleep. Such palpitation is due to an increase in the blood pressure in the arteries, which requires a more energetic ventricular contraction to overcome it. Here again palpitation is the active evidence of debility. Be it always remembered that such palpitation is very frequently found accompanied by the first form—that induced by effort. Palpitation is easily induced by effort in the enlarged and dilated hearts of women of middle age, at the change of life, who are the subjects of lithiasis. The diagnosis turns on the fact that the attacks of palpitation are not solely

caused by effort, but arise under other circumstances in this second form. As bearing on this second form of palpitation, I may quote a remark made by a patient, a Westmoreland peasant, the subject of old-standing mitral regurgitation (25 years).

He said, in a letter to me, that he often suffered from attacks of palpitation, but that for three weeks he had suffered from a cold. "It is curious," he remarked, "that I never suffer from palpitation then." The change in the square sphygmographic tracing of chronic lithiasis under a cold is significant. The square head is changed for the pointed apex of pyretic conditions. Consequently, we can understand that this crippled heart never palpitated when the blood pressure in the arteries was lowered by a cold. This observation is pregnant with the most valuable information as to the relations of palpitation to the blood pressure in the arteries, and to the amount of obstruction to be overcome by the ventricular systole. Then (3) palpitation is found under a totally different set of circumstances, where it is a neurosis, and where in every way it differs from the forms just described. The heart's action is carried on by the cardiac ganglia, and regulated by different nerve connections, as was seen in Chapter I. Disturbance in the working of the heart may, and very often does, arise in these cardiac ganglia. The discharges from these rhythmically discharging centres may be increased in rapidity and force, and are accompanied by corresponding ventricular contractions. Here palpitation may fairly be described as overaction of the heart. As may readily be conjectured, such a neurosis is most commonly found in woman, whose emotional nervous system renders her very liable to neuroses. Slight exciting causes, as fright, excitement, or other emotion, set up violent action of the heart, which beats as if it would burst open the thoracic parietes over it. Patients with chorea and Graves's disease have usually such palpitation; so have some women who have neither of these affections. In one instance known to me, a mother and daughter have each a heart of this character. In the further consideration of the neurosal affections we shall see that such palpitation may be reflex—due to some irritation elsewhere, as in the uterus or ovaries; or it may be a primary neurosis. Such palpitation is often found along with an emotional temperament in young or

delicate women, and is not accompanied by any corresponding perturbation in the arteries; the radial pulse being quiet and regular during the time of the attack.

Then comes irregularity of the heart. By this is meant irregularity in time, not irregularity in volume, the latter being pathognomonic of mitral disease, and of conditions of marked dilatation of the left chamber of the heart. Irregularity is a brief ventricular hesitation, which may be explained by a slight digression. The sensation of distension on the internal surface of the heart received by the cardiac ganglia is the exciting cause of the rhythmic discharge which sets up the cardiac systole. While there exists nominally a balance betwixt the sensation of distension and the excitability of the cardiac ganglia, controlled by the vagus fibres, this may be disturbed by certain conditions. When these centres are exhausted, a more prolonged excitation may be requisite to excite the discharge, and thus a halt be produced; or there may be a species of storing up of energy, as this brief pause is not rarely followed by a more determinate blow, a ventricular systole more powerful than the other cardiac contractions. But quite commonly this irregularity is a mere trick of no significance. We saw that the muscular wall of the heart consisted of seven layers of fibres which have been unrolled, and it seems quite possible that, in those cases where it is a mere peculiarity, that at times the systole, instead of commencing at fibre number 1, may tarry until a set acting later normally, and so commence from number 3 or 4. This last form of irregularity is not affected by exertion; the first form is so affected, and is increased in frequency by effort.

B. W. Richardson has likened this irregularity to "a smith who, striking at a forge a number of strokes in rhythmical succession until tired, changes the action for a moment to give a more deliberate and determinate blow, and then rings on again in regular time." It has always struck the writer as most resembling the "change" of a horse's feet when cantering; a momentary pause, during which the other foot is put foremost, and then on again. The heart beats away rhythmically, and then comes suddenly a sort of roll-over of the heart, and then on again. This is quite indescribable by words, but the reader who has once

experienced this curious phenomenon will recognize at once what is meant. Frequently such irregularity is found as the patient enters the consulting-room, but after a brief time can no longer be heard.

Where any doubt exists as to whether the irregularity is due to a mere nerve disturbance, possibly of perfect balance betwixt the restraining power of the vagus and the cardiac ganglia, or a disturbance of balance betwixt the opposition offered by the blood to be driven and the muscular power to drive it, the question admits of ready solution. Let the patient make an effort. Over the mere nerve disturbance this exercises no influence; but when it is connected with cardiac debility this increase in the demand upon the heart has a very perceptible effect in increasing the amount of irregularity. Frequently this pause is preceded by three or four rapid feeble contractions; then the pause, with or without a more determined systole, immediately following; then come some normal strokes, followed by the rapid feeble strokes and the pause. This condition indicates grave debility, and is found along with pronounced dilatation, with or without mitral disease. It is well communicated to the radial artery, which, if atheromatous, will exaggerate these changes. When such a condition is permanent, palpitation is readily induced by effort.

The last and most serious evidence of cardiac failure is intermittency, *i. e.*, arrest of ventricular systole over one or more auricular contractions. This prolonged halt of the heart has been the subject of much investigation, especially since the brothers Weber first discovered that irritation of the vagus nerve will arrest the cardiac contractions. Slight irritation slows the heart's action; more pronounced irritation arrests it. It has been stated that in intermittency the ventricle halts over an entire cardiac cycle, and that a second auricular contraction is required to set the ventricle in action. Two rats had their chest-wall opened, and it was found that as the animals died the ventricle began to intermit. At first a second auricular contraction was sufficient to excite the ventricular systole; but later on even the second auricular contraction was insufficient to excite the contraction of the ventricles, which halted for a third and even a fourth auricular contraction; but at last the auricular contraction passed swiftly

on to the ventricle, and the cardiac systole was completed. That intermittency of the heart is a ventricular halt over one or more auricular contractions may fairly be asserted. It may be that the increased internal pressure upon the ventricular surface produced by these increased auricular contractions is necessary to excite contraction in the ventricles. In practice, then, we often find intermittency to indicate ventricular arrest from adynamy. In auscultating a heart with fatty degeneration, not rarely the pause of the lagging ventricle suggests the fear that the arrest is going to be final. When found along with other evidences of heart failure this long ventricular halt is of the gravest import; and when found with a diminished first sound, tortuous, atheromatous arteries, panting respiration, a cloudy cornea, and a genuine arcus senilis, is significant enough. It tells that the muscular structure of the heart is undergoing degenerative changes, no matter whether hypertrophy has once been present or not, though commonly enough it has been pre-existent. The effect of effort here is not to induce palpitation, for the heart is not equal to it; palpitation is an effort beyond it. Effort produces longer and more frequent halts.

Intermittency of grave prognostic omen is not, however, always so associated; and the two most marked instances in the writer's experience were both connected with aortic obstruction. In each the intermittent halt extended over no less a period than four cardiac cycles. The sensations of these patients during these periods of ventricular arrest were of the most horrible description; the terror excited was visible in their countenances, and was quite in accord with the description given by Romberg in his "Diseases of the Nervous System" (vol. ii) of a patient in Professor Heine's wards in Vienna, where the heart stood still over a period of six normal beats, the rhythm being good at other times and during the intervals. (In this case there was a tumor involving the vagus.) In these cases of ventricular arrest the horrible sensations are due to the brain being temporarily deprived of its normal amount of arterial blood. Intermittency is, then, an evidence of structural degeneration at times, while at other times it may be present where a strong ventricle halts before a decided obstacle to the blood flow.

Again, intermittency may be a purely nervous phenomenon. It may be due to some irritation in the vagus, as in the case just mentioned. The famous Czermak, of Prague, had a growth near his vagus on one side, and by pressing it could arrest the heart's action more or less completely according to the amount of pressure. Such cardiac arrests may be attained by an effort of will, as in the well-known case of Colonel Townsend, who, it is often stated, killed himself by such arrest of his heart; or it may be occasioned by some disturbance of the nerve-balances of the heart, as Dr. B. W. Richardson has so ably demonstrated. He has shown that it may be induced as the result of shock, as in shipwreck; by grief, or by anxiety; it may be found at birth, and afterwards disappear; it has also been observed in animals. Cases where intermittency has existed for years have presented no morbid change to be detected after death. (The subject will be referred to at greater length in Chapter XIV.) Dr. Richardson goes so far as to divorce intermittency from organic disease of the heart altogether; and though it is not possible to go this length with him, still he has done enough to amply demonstrate that no practitioner is justified in giving an opinion that heart disease exists, on the evidence of intermittency only. If supported by other data, then intermittency acquires significance, until it may possess a high diagnostic and prognostic value. But alone it may mean nothing. Yet such hasty medical opinions have been hazarded; and I remember well a hale, stalwart north country yeoman, whose peace of mind was ruined by a rash medical opinion. He was a healthy strong man years after this hasty medical adviser was in his grave; but the opinion formed so unjustifiably had made so strong an impression on him, that no amount of assurance of his health could free him from the terrible incubus of the idea.

Intermittency may be a symptom of cerebral disease, as cerebritis, tubercular meningitis, or hydrocephalus; and when it occurs is of bad omen. (Marshall Hall.)

Finally, we have irregularity and intermittency as the common prodromata of death. In many cases we form our opinion of how long it will take for the sands of life to run out, from the character of the pulse. When the pulse becomes irregular and then commences to intermit, we know the end is not far distant.

In all serious acute disease such changes in the pulse are regarded as highly significant, and are viewed with great anxiety. In such intermittency the right ventricle is the chamber chiefly embarrassed, and especially so in disease of the respiratory organs. Commonly enough in dead-houses, the right ventricle is found distended with blood, while the left ventricle is firmly contracted. No doubt many muscular fibres are common to both ventricles, and so the chambers keep time with each other; but often it is obvious that the right ventricle is struggling away with difficulties, when the left ventricle is in no way taxed. Proof that such unrhythmical action is connected with the right ventricle is furnished by the fact that the halt in the right ventricle may be made out by auscultation before it is manifest in the radial pulse. How far irregularity and intermittency of the pulse is connected with right-side embarrassment in some cases, has not been sufficiently worked out. In cases of cardiac asthma, where the right ventricle is distended without the left being embarrassed, and in that right-side distension produced by the long-sustained efforts of divers, gymnasts, runners,* and others where the right ventricle becomes strained, irregularity and intermittency of the radial pulse are obviously connected with the right heart; and these phenomena are much more distinct to auscultation than to the finger on the pulse.

Aid derived from the Arterial System.—Much useful information is furnished by observations made on the arterial system, in addition to examining the heart. The elastic arterial system often gives out very accurately the impression made upon it by the ventricular contraction. The rapidity and the rhythm especially are thus faithfully indicated. It is customary to feel the patient's pulse ere proceeding to make a careful physical examination of the chest; and to the educated finger the pulse often tells much more than the mere time and rhythm; it will often tell that there is valvular disease present. Not only that; but which valve is affected, and whether the disease be obstructive or regurgitant. Consequently, it is well to cultivate acquaintance with the pulse; as it will commonly give valuable direction to the physical exam-

* See Clifford Allbutt, On Distrain of the Heart, 1872.

ination of the chest. The frequency of the pulse should be noted, especially as to the effects of the respiration upon it; inspiration slowing it, while expiration accelerates it. At times, too, the real rate of the pulse is obscure, it appears slower than it is, or faster than it is, when carefully counted with the watch. Thus there is "the quick slow pulse," and "the slow quick pulse." Great frequency may be suggestive of nervousness, and passes away as the examination progresses; consequently in great frequency it is well to take the pulse at the commencement, and again at the end of the examination. Persisting great frequency is not a pleasant fact; it may indicate great debility, or irritability in the heart. Then the pulse-rate may vary, and the rhythm be different at short intervals of time. Unusual slowness may be neurosal, or it may indicate fatty degeneration; at times it indicates that only so many of the ventricular contractions reach the radial artery. The character of the pulse is more indicative usually than the time or rate. If strong and the radial artery is compressible, it indicates hypertrophy with high arterial tension. When weak and compressible, it indicates fatty degeneration, dilatation, or debility, especially if at the same time it be unrhythmical. It is under such circumstances somewhat abrupt. It is drawn out or prolonged in hypertrophy with arterial atheroma, as in Bright's disease. Then it may be "full" without being hard and incompressible. This indicates vasomotor dilatation, and is found in pyrexiæ and thoracic inflammations; while at other times the pulse is small and "wiry" and incompressible, as in abdominal inflammation, and in some cases of dropsy which are renal as well as cardiac. In hysteric attacks the same kind of pulse is found. Then the pulse may be readily compressible and obliterated by pressure, especially during the arterial diastole; such a pulse indicates cardiac debility. While a pulse of opposite character, which can be rolled under the finger during the diastole, indicates high arterial tension, that is arterial fulness, with an hypertrophied left ventricle. Whenever atheroma is present, it exaggerates the pulse and gives it a feeling of strength which is unreal. Consequently, in a person with atheromatous arteries, the pulse has a fictitious strength which is often misleading. In a near relative of the writer's this condition of arteries existed, and even during periods

of illness and depression, when tonics and stimulants were really needed, the strong radial pulse, from an hypertrophied left ventricle and atheromatous arteries, almost seemed to indicate venesection. At times of greater depression, as after acute diarrhœa, a distinct intermission could be felt apparently quite inconsistent with such a pulse, yet indicating correctly the passing condition. This effect of atheroma in exaggerating the pulse is well worth noting and remembering in practice. It is well, too, to accustom the eye to observe the temporal arteries, which often furnish valuable information. In elderly persons, the temporal arteries are often tortuous and rigid, indicating atheromatous changes, and suggesting chronic renal disease. The power of atheroma to exaggerate the impression made by the pulse giving a heave to its impulse, can often be noted in the temporal artery when, sinuous and rigid, it becomes distinctly elongated as well as widened by the ventricular systole. Sometimes this artery is small and rigid; in others, it is thick and soft; indicating modifications of the atheromatous process. In many cases each beat of the pulse can be seen in the temporal artery, if placed where the light falls distinctly upon it. In younger persons the temporal artery may be conspicuous and tortuous; and such is often the case where the habits are studious. At other times, a yellow parchment-looking skin, seeming to be tightly stretched over the forehead, covers a tortuous temporal artery, which is very conspicuous; here there is a strong suspicion of syphilis, which in some persons seems to settle on the arteries more than any other part.

Then the radial pulse is modified by disease of the valves of the heart; often sufficiently so to indicate whether the mitral or aortic valve is the seat of disease, and of what form of disease, or even at times that aortic regurgitation is accompanied by mitral regurgitation. In aortic obstruction the pulse is small, steady, and sustained, and usually rhythmical, though when the ventricle is failing intermittency may be present. In aortic regurgitation the pulse is very significant. Here a ventricle enlarged and thickened throws an abnormally large bulk of blood into the arteries, much of which escapes backwards through the imperfect aortic valves. Consequently, though the arteries are greatly distended, they quickly recoil, and the pulse is cut off, as it were.

It feels as if "balls of blood were shot under the finger," and has been described as "splashing," or as a "water-hammer" pulse. It has also been termed a "throbbing" or "collapsing" pulse, from its sudden "fall back." Such a pulse is very visible to the eye, not only in the temporal but in other less conspicuous arteries. It is modified by the presence of mitral regurgitation, as will be described in Chapter VI. In mitral obstruction the pulse is small, but regular and rhythmical, though George Balfour, of Edinburgh, holds that the pulse of mitral obstruction is very irregular, in which he is in disagreement with London observers. In mitral regurgitation the pulse is irregular in volume, as sometimes more blood escapes through the insufficient mitral valves than at other times. It is also irregular in time when the left ventricle is dilated, and still more when the heart is failing in the later stages. In those cases where there is both mitral obstruction and dilatation the pulse is modified exceedingly; it is small, weak, and more or less irregular. In all cases of right-side disease, especially in tricuspid regurgitation, the radial pulse is ever weak. The left side of the heart can only transmit to the arteries the blood coming into it from the pulmonary veins; and if there be any leak in the right side pumping apparatus, the amount of blood passed into the pulmonic circulation is comparatively small, and thus the arteries are imperfectly filled, no matter how healthy or efficient the left ventricle may be. The radial will often tell us that a strong heaving cardiac impulse is due to right side enlargement, and not to hypertrophy of the left ventricle, by its being disproportionately small to the impulse. Here the right side enlargement is compensatory to some obstruction lying betwixt it and the radial pulse, as some disease of the lungs or left heart, so that the radial pulse is not affected. At other times a strong and vigorous radial pulse will tell of a stout and active heart when the cardiac impulse cannot be felt, and the sounds are heard but imperfectly, as where rigid costal cartilages coexist with emphysematous lungs, especially the anterior edges. Here the mistake has been made of diagnosing a weak, fatty heart, from not observing the character of the pulse carefully enough. In conditions of anæmia the pulse is necessarily small, and cannot reflect faithfully the condition of the heart.

Murmurs, too, are heard in arteries. The murmur of aortic obstruction may be carried some distance by the arterial current. Aneurisms produce murmurs (which will be described further on); tumors, or abscesses over arteries, give rise to murmurs, as will also the pressure of the stethoscope. Arteries, where numerous, possess a souffle, as the utero-placental souffle, and that of the thyroid gland in Graves's disease, or cirsoid aneurism. Attempts have been made to auscultate the encephalic arteries with indecisive results. The wide-dilated aorta, with a roughened interior, found in some cases of atheroma, with or without aortic regurgitation, but commonly along with it, gives rise to a murmur which can be heard distinctly, as well as to a thrill, which can often be readily felt by pushing the finger into the manubrium sterni while the patient thrusts the head forward. The huge long heave with the thrill is often very distinct. In atheromatous conditions of the arteries the brachial artery is often cordlike, so as to be rolled easily under the finger, and has a distinct thrill.

A subclavian murmur can often be found, especially in working men who use the arms much, which suggests aneurism. It is thought to be due to the action of the subclavius muscle; at other times it is due to disease of the apex of the lung.

Aid derived from the Venous System.—Valuable information is often furnished by observing the venous system in disease of the heart. In conditions of congenital disease there is usually a condition of blueness or cyanosis, which is pathognomonic thereof. At other times, in heart failure, there is an approach to this condition, and the features are blue, especially the lips, and are also blurred in outline from venous fulness. This is found with dilated chambers, and usually with mitral disease. The most marked blueness of face and hands ever seen by the writer was in a man where chloral-intoxication was added to mitral regurgitation. In heart failure, where the central pump does not lift the blood out of the veins efficiently into the arteries, the veins are too full of blood; or, as the Germans say, the blood is lying too much on the venous side. The venous system, lying betwixt the systemic capillaries and the pulmonic circulation, has the right heart for its pump, aided by some auxiliaries, as inspiration and the muscles of the limbs. Failure of the right heart, then, means

an imperfect emptying of the veins and venous congestion. When the right heart is dilated, and there is tricuspid regurgitation, then the veins are distended with blood. They hold not only the blood coming into them from the systemic capillaries, but also that which regurgitates through the leaking tricuspid at each systole of the right ventricle. The veins are arranged to permit of a slow current within them, with a very low internal pressure, and when they are thus subjected to such a distending force as the contraction of the right ventricle, they readily dilate and their valves become insufficient. Consequently, when the tricuspid valve no longer effectually secures the veins against the action of the right ventricle, the veins of the neck become dilated, and pulsate with the contraction of the right ventricle. This may occur to a great extent, however, without a tricuspid murmur being audible; indeed, the murmur is comparatively rarely heard. Tricuspid regurgitation is commonly accompanied by rhythmical pulsation of the liver, a diagnostic point to which the Germans pay great attention. The regurgitant wave passes down the vena cava into the veins of the liver, and causes that viscus to pulsate with the contraction of the right ventricle. The theory of Wilkinson King of "the safety-valve action of the tricuspid," finds no acceptance with German authorities. Then the veins become full, when, from temporary causes, the right ventricle is embarrassed and distended as in asthma, or in the attacks of cardiac dyspnœa, which so often occur with mitral disease, especially towards the end of the case, for the tricuspid is the least perfect of all the valves of the heart. Venous fulness may, however, be due to a tumor pressing on one or both venæ cavæ, to a thrombus, or an aneurism in the thorax. It is seen after a violent fit of coughing, especially if the heart be weak. "Care must be taken not to mistake transmitted arterial pulsation for venous." (F. Roberts.) A thrill may accompany venous pulsation.

In conditions of anæmia a distinct "hum" can often be heard in the veins of the neck, especially at the junction of the internal jugular and the subclavian. It is known as the *bruit de diable*. It is continuous and uninterrupted, though heard with greatest intensity during the ventricular systole. It is commonly accompanied by a hæmic murmur at the pulmonary orifice. Inter-

rupted murmurs are very rare. The cause of venous murmurs is yet in dispute; though many ingenious hypotheses have been raised, none as yet has been accepted.

Veins often partake of general atheroma, and are full, round, and more or less rigid, not collapsing on the finger being placed on the distal side of them. This is seen most marked in the veins of the forearm, and in the coronary veins, which last are often large, tortuous, and rigid, when the foramina Thebesii are compressed by the distension of the right auricle. The late Professor Laycock, of Edinburgh, used to lay stress on the dilatation of the pericardial veins in cases of heart disease.

By lifting the arm the pulse is nearly lost in anæmia. In mitral disease the pulse may be rendered more irregular by lifting the arm. By so doing the collapsing pulse of aortic regurgitation is made more distinct. If a pulse at once small, feeble, and irregular, as well as collapsing, has these characteristics rendered more marked by holding up the arm, "then we have to do with a double lesion, a mitral, and also an aortic regurgitation." (Balfour.)

The effects of effort in increasing the internal pressure within the cardiac cavities can often be utilized in forming a diagnosis in obscure cases. The plan of getting the patient to walk quickly round the room, and then examining him again is in vogue; but any plan of exciting effort is as effectual. When effort taxes the heart palpitation may be set up, indicating debility in the heart, which beats rhythmically enough when the patient is quiet, or it may bring out a mitral murmur, telling that the mitral vela are injured and contracted to some extent. During quietude they are equal to closing the ostium, but when the cardiac chambers are somewhat distended, the ostium enlarges therewith, and regurgitation, producing a murmur, is set up. This is in accordance with what was said in the first chapter about variations in size of the cardiac chambers.

While the physical signs of disease of the vascular system are of very great value, they should not be overestimated, as they recently have certainly been. They are often inferior in value to the information furnished by the patient's subjective sensations, and often these latter can alone enable us to distinguish betwixt

organic disease and conditions which simulate them closely. It is often much easier to detect a murmur or irregularity of action in the heart than it is to appraise their significance and value. From this point of view heart disease is not a matter to be always settled by the stethoscope, however valuable that instrument may be. There is no such thing in this world as unalloyed good, and the present devotion to physical signs and instruments of precision has led to a comparative indifference towards, and neglect of, that broad view of a case, with all its points included and fairly appraised, for which the older school was conspicuous, and it may be asserted truthfully enough that increasing familiarity with the stethoscope and other instruments has too often been accompanied by a corresponding indifference to information afforded from other sources of equal or even greater value. The diagnosis in disease of the heart is not the end but the beginning of the examination, and its accuracy is of value, not as a clever feat of intellectual legerdemain, but only in so far as it enables the physician to comprehend the disease, and strengthens his hands as to the appropriate treatment. This view of the value of a correct diagnosis is not always sufficiently kept in mind.

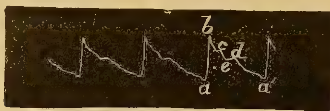
The more accurate observation of the pulse has lately been rendered available by means of the sphygmograph, and my friend, Dr. Balthazar Foster, whose work with that instrument is well known, has furnished me with the following notes:

"Defects in the mechanical perfection of the heart, as well as modification in its contractile vigor, have long been recognized by changes in the radial pulse. The pulse signs of heart disease have consequently always had a high value. The application of the sphygmograph to clinical inquiry has given these signs an increased importance by revealing their precise features, and the comparative modifications of pulse form under different morbid conditions, and at different times.

"The healthy pulse curve (Fig. 16), registered by the sphygmograph, consists of a line of ascent (a to b), a summit (b), and a line of descent (b to a'). The line of ascent is very slightly oblique, and terminates in the summit wave (b). The line of descent is broken by two secondary waves (c and d), one of which follows closely on the summit wave, and in all probability is the

true pulse wave, the summit wave being artificially separated from it by the instrument. This wave (*c*) is called the wave of impletion or tidal wave, and is followed by a fall in the line of descent, immediately after which rises the large wave (*d*) called

FIG. 16.



Pressure 31 grams.

the dicrotism. The dicrotism is mainly produced by the rebound of the blood column from the closed aortic valves. This closure is indicated in the trace by the notch (*e*) (aortic notch), which precedes the dicrotic wave, and corresponds to the heart's second sound. The pulse curve may, therefore, be separated by this notch into its systolic and diastolic portions; the line of ascent, the summit wave, and the line of descent as far as the aortic notch (*a* to *e*) correspond with the ventricular systole; the dicrotism and the remainder of the line of descent (*ε* to *á*) to the ventricular diastole. Slight undulations following the dicrotism are occasionally seen, but these have no special significance.

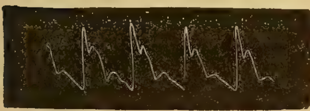
" Modifications in these several parts, as well as peculiarities of rhythm, and changes in form and volume, are recorded by the sphygmograph, and from them we learn not so much to diagnose the special cardiac lesion, as to gather information respecting the general state of the circulation, and thereby gain hints for prognosis and indications for treatment.

" In hypertrophy of the left ventricle, for example, each pulse curve in trace Fig. 17, shows by its vertical line of ascent and its lofty summit wave, the suddenness and vigor of the systole. The well-marked tidal wave tells of the filling of the arteries, and the distinct dicrotism of the rebound of the blood column from the perfectly closed aortic valves, and its free passage onwards through elastic vessels not too full.

" In Fig. 18 we see again the pulse trace of left ventricular hypertrophy under the different conditions of high arterial tension, due to the obstructed capillary circulation of chronic renal dis-

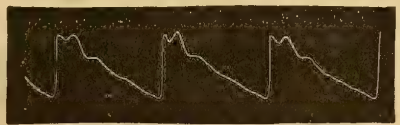
ease. Here the lofty summit wave is surpassed by the exaggerated tidal wave, which tells of strong ventricular contraction and full bloodvessels, and betrays itself to the finger by the fulness and

FIG 17.



Pressure 48 grams.

FIG. 18.



Pressure 85 grams.

hardness (incompressibility) of the pulse. The poor dicrotic wave and the slow-falling line of descent show how little rebound the tense blood column allows on the closure of the aortic valves, and how slowly the distended arteries pass their blood through the unyielding capillaries.

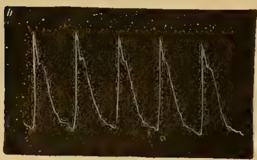
" In a pulse of low arterial tension these features are all changed, the tidal wave is blended with the summit wave, or barely marked, and the dicrotism stands out boldly, as may be seen in the tracing of the empty pulse of mitral regurgitation, Fig. 25.

" In aortic regurgitation the pulse curve always presents striking characters. The lofty line of ascent, and the hooked summit, which were at one time regarded as peculiar to this condition, are really often met with in neurotic persons with functional heart disturbance. A sudden and fairly vigorous systole, in a state of low arterial tension, develops these features. The true peculiarities of the pulse of aortic regurgitation are found in the line of descent, which falls suddenly, and is broken by little or no attempt at a dicrotic wave. The sudden fall of arterial tension, indicated by the line of descent, results from the rapid emptying of the arteries, partly by reflux through the incompetent valves, and partly by the rapid passage of blood onwards through the arterioles. The maxima and minima of tension quickly succeed one another, and the dicrotic wave, which should mark the rebound of the blood column from the aortic valves, is spoiled or lost by their incompetency.

" In cases of very free regurgitation, as in Fig. 19, these peculiarities are well seen. The lofty upstroke, the pointed summit wave, the lessened tidal wave, the rapid fall of the line of descent,

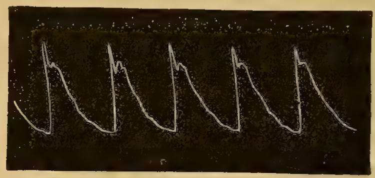
and the almost total abolition of the dicrotism, which only shows faintly at the end of the line of descent, may all be noted. In this case there was very free regurgitation, from tearing and retroversion of one valvular segment and shortening of the others.

FIG. 19.



Pressure 36 grams.

FIG. 20.

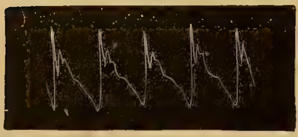


Pressure 40 grams.

"In cases in which there is thickening of the valves and atheroma of the great vessels, the tidal wave is often more marked, but the free regurgitation still spoils the dicrotic wave (Fig. 20). In other cases in which the valves are studded with vegetations, and the reflux is less free (Fig. 21), all the waves of the pulse may be well marked, and the dicrotism, though short of its full development, may still show distinctly.

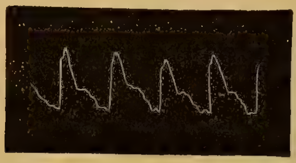
"In aortic obstruction, or stenosis, the blood is discharged more slowly through the narrowed aortic orifice; the line of ascent is consequently modified, and the summit wave, no longer produced by the shock of a quick systole, loses its point. In some cases the line of ascent is so far changed as to be oblique, the summit of the trace is formed by the tidal wave; and the dicrotism, from the slow ventricular contraction and necessarily slow aortic recoil, is badly marked, or lost in the gradual fall of the line of descent. In Fig. 31 (a trace taken under other conditions) we have a very good example of these modifications.

FIG. 21.



Pressure 51 grams.

FIG. 22.



Pressure 24 grams.

"In other cases the line of ascent, instead of being oblique, is broken (Fig. 22), the break indicating the position of the summit

wave or shock element; the real summit is, in such case, formed by the tidal wave. The gradual, slow, but forcible contraction of the ventricle, makes the summit wave less perceptible, and the tidal wave forms the apex of the pulsation. The dicrotism in these cases is marked, but not fully. In this case there was rigidity of the aorta, as well as narrowing of the aortic orifice, from thickened and degenerated valve cusps.

"In mitral regurgitation the pulse curve shows many varieties, but its main features are irregularity and low tension. The individual pulsations may often retain the normal form, but the varying quantities of blood discharged into the aorta by each systole, and the hurried, irregular heart action, give the trace a very ataxic character. These features are most marked when the condition of asystoly is present (Fig. 23). This tracing was taken from a case of free mitral regurgitation, due to rheumatic inflam-

FIG. 23.

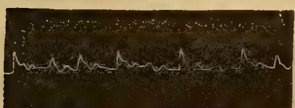
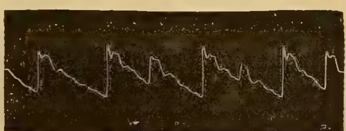


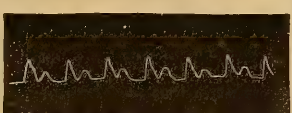
FIG. 24.



Pressure 42 grams.

mation of the valve curtains. The same patient, after fourteen days in the hospital, under treatment by digitalis, had the pulse recorded in Fig. 24, in which we see the good effects of the drug in curing the asystoly and restoring a more normal balance of the circulation, by its toning action on the heart muscle. The irregularity of the pulse is still present, but many of the pulsations are almost normal in form and vigor.

FIG. 25.

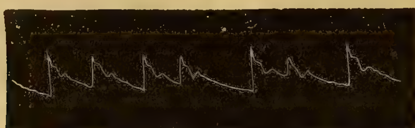


"In some cases of mitral regurgitation the pulsations, while not much altered from the healthy type, are increased in frequency, and are incomplete or starved in volume from the small charge of blood received from the ventricle at each systole. In Fig. 25

we have a pulse tracing showing these characters, and the post-mortem examination, while it disclosed adherent pericardium and great dilatation of left ventricle, showed the mitral valve so shrivelled and incompetent, as to explain readily the small arterial wave by the free mitral leakage at each systole.

"In mitral obstruction or stenosis, the lesion has from its seat no direct influence on the discharge of the blood wave into the aorta, and consequently does not necessarily modify the form of pulse curve. The narrowed mitral orifice, however, lessens, or tends to lessen, the volume of each ventricular charge, and therefore we have in many cases, while the patient is at rest, a perfectly regular pulse, but of low tension. When, on the other hand, the heart is taxed by any muscular effort, or by respiratory troubles, the pulse becomes irregular. In advanced cases this irregularity is the rule, and is often of a very extreme form. Ordinarily the irregularity

Fig. 26.



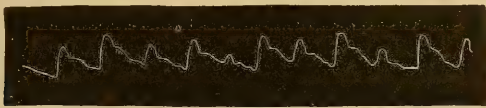
Pressure 38 grams.

is less developed, being similar to that seen in Fig. 26, a tracing taken from a man whose adherent valve curtains formed a button-hole orifice only admitting the top of the forefinger. The tracing was taken when he was fairly well. The pulsations, it will be observed, are all unequal, no two succeeding ones being precisely alike, but each still presenting all the elements, though modified, of a healthy pulse curve. The pointed summit wave, and well-marked dicrotism, tell of the low arterial tension, and the break, as of a missed pulsation in the middle of the trace, is another feature often seen.

"Valvular defects of the right side of the heart do not betray themselves by any special modifications of the pulse curve; their only influence when primary, is by the lessened arterial tension which they induce. The left heart is less perfectly filled, and

the pulse tension is consequently lower. The trace, Fig. 27, shows a form of pulse often recorded in cases of general dilatation of the heart cavities with tricuspid regurgitation and atheromatous bloodvessels.

FIG. 27.



Pressure 44 grams.

"The oblique line of ascent, the occasional abortive systole, the rounded summit in which are blended the summit and tidal waves, all indicate the feebleness of the ventricular systole. The inelastic state of the great vessels shows itself by the rounded summit wave, by the early occurrence and by the feeble development of the dicrotism.

"In aneurism the modifications of the pulse are closely connected with the seat of the tumor, its size, and the elasticity of its walls. In aneurism affecting one of the larger arteries on which the pulse can be taken beyond the aneurism, the sac, if elastic, may act like an elastic bag on a schema of the circulation, and completely modify the form of blood movement, reducing the pulse curve to a simple form where the gradually sloping lines of ascent and descent are almost equal. This extreme alteration is seldom observed, but modifications in this direction occur when an aneurism produces a general dilatation of a large artery like the subclavian or the innominate. The finger in such cases may fail to feel any pulsation, while the sphygmograph records only a faint curve. (*Vide* 'Foster's Clinical Medicine,' p. 295.)

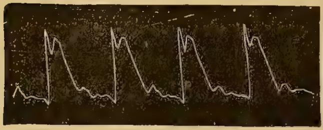
"In aneurisms of the thoracic aorta modifications occur in the force and volume of the pulse, in the development of the dicrotism, and in the persistency of differences between the two radial arteries.

"The relative force and volume of the radial pulses are not constantly affected by aneurisms of the first portion of the aorta, unless the lesion implicate the mouth of a main branch. The vigor of the cardiac systole usually gives the pulse considerable volume, and the size of the sac and its situation do not often

modify the line of ascent or summit. The radial pulses are, however, very often dissimilar. There is a want of parallelism in the beat of the vessels and in their tracings. The latter often show this when the finger fails to detect it. The want of similarity is generally seen in the line of descent, in the altered relative size and position of the secondary waves, the dicrotism more especially.

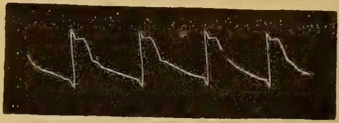
"Pulse traces, Figs. 28 and 29, are taken from a case of aneurism of the ascending aorta complicated with aortic insufficiency. The aneurism left the orifice of the innominate artery free. The right pulse shows, compared with the left, a somewhat increased force and volume, but these points are less significant than if they had occurred in the left radial, which is normally the weaker of the two. The really important points of difference, and these were persistent, are to be seen in the secondary waves. The tidal

FIG. 28.



Right Radial. Pressure 68 grams.

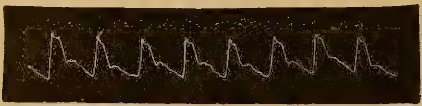
FIG. 29.



Left Radial. Pressure 52 grams.

wave, though well seen on both traces, shows a preponderance on the right, while the fall of the line of descent is much more sudden. The dicrotism, too, is slightly marked in the right pulse curve, while it is almost lost in the left, being absorbed by the aneurism. The vibratory waves which follow in the right trace are eddies produced by the sac's contraction, which failed to reach

FIG. 30.

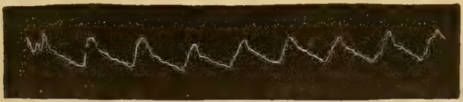


Right Radial. Pressure 42 grams.

the left radial. In Figs. 30 and 31, taken from a patient in whom there was found a large aneurism of the arch, both pulse

traces are affected. The sac from its position modified the blood waves entering the innominate and the left subclavian. In both pulse curves the force and volume seem to be lessened; the sloping line of ascent in the left tracing shows how the sac obstructed the flow of blood into the left subclavian; the same feature is seen in a less degree in the right pulse. The dicrotism is lessened on both sides, though much more so on the left; some of the rebound of the blood column reached the right pulse, but the sac intercepted it on its way to the left. The decided changes in the form of the left tracing in the oblique line of ascent, the loss of the summit wave and obliteration of the dicrotism, pointed decidedly to the aneurism being so placed as to obstruct the free flow of blood into the left subclavian.

FIG. 31.



Left Radial. Pressure 28 grams.

"In aneurisms of the abdominal aorta the radial pulse traces give few, if any, direct indications. In some cases waves of oscillation generated by the sac modify the line of descent of the left radial, and also sometimes affect the right. The dicrotic wave has been found exaggerated in size by receiving a reinforcement from the contraction of the sac. Permanent differences may also be found occasionally in the tracings of the femoral or posterior tibial arteries. When the sphygmograph is applied to the radial pulse pressure exerted on an aneurismal sac will be indicated by changes in the tension signs of the pulse, and thus indications for diagnosis may be obtained. The application of the sphygmograph over a pulsating tumor by recording the finer features of the pulsation will often also aid in diagnosis. The more perfectly the tracing so obtained exhibits the main elements of the movement of the larger arteries, the more it points to aneurismal dilatation."

CHAPTER IV.

THE CONSEQUENCES OF OBSTRUCTED CIRCULATION—THE SUBJECTIVE SYMPTOMS OF HEART DISEASE.

THE various pathological outcomes of obstruction of the circulation by disease of the heart are numerous, and may be discussed together profitably. By taking them *en bloc*, their consequential relations to the changes in the heart will be all the more obvious and distinct. They have received much attention from the Germans, who lay great stress upon the "rückwirkung," or "backworking," *i. e.*, the pathological changes set up by the obstruction of the circulation at the heart.

As long as the mitral valve remains competent, we see little of this "rückwirkung." In aortic disease there are no complications involving the pulmonic circulation and the venous system, until the mitral valve becomes secondarily implicated. In aortic regurgitation the arterial system becomes involved. The huge ventricle found therewith distends the arterial system violently, aud the arteries generally become atheromatous, the aorta not uncommonly being dilated as well as covered with atheroma. Rupture of a cerebral artery is not uncommon in aortic regurgitation. In aortic stenosis the narrowing of the aortic orifice prevents any such effect; there the enlarged ventricle is compensatory to the stenosis. In aortic regurgitation the matter is more complex, as will be seen in the succeeding chapters, VI and VII.

When the mitral valve is the seat of disease, then backward changes are set up—changes far-reaching and very important. It is of little consequence whether the mitral disease be primary or secondary to aortic disease. Equally of little consequence is it whether the mitral disease be stenosis or insufficiency. (Except that Dr. Broadbent thinks mitral stenosis is more liable to be followed by effusion from the peritoneum, the consequences (Nachfolgen) are the same.)

The first consequence of obstruction or damming at the mitral

orifice is fulness and distension of the pulmonic circulation. This is followed or rather accompanied by enlargement of the right heart, the cavities become enlarged while their muscular walls are thickened. By this conservative nutritive change, the blood pressure in the pulmonic circulation is heightened, and the pressure in the pulmonic veins presents a greater obstacle to the backward flow on regurgitation; while it impels the blood more energetically through the narrowed ostium in stenosis. The large pulmonary capillaries permit of right-side hypertrophy exercising a considerable influence upon obstruction of the circulation at the mitral orifice.

This fulness of the pulmonary vessels leads to distinct pathological consequences of its own. The vessels themselves become altered, and atheromatous changes are set up in their walls (Gerald Yeo). The pulmonary artery may become incrusted with atheroma, and even the pulmonary valves may be somewhat implicated. But as is shown by a case of Wynne Foot's in Chapter VII, the changes may be such that they can scarcely be called disease; they are nutritive changes, but not necessarily morbid. The compensatory growth changes in the right heart in cases of pulmonic obstruction do not necessarily mean valvulitis and atheroma; but usually atheroma of the pulmonary artery is induced where there is a powerful right ventricle, and the case runs a chronic course. The pulmonary valves have rarely more than a tiny festoon of atheroma on their cusps; though the pulmonary artery and its branches become extensively atheromatous, and this atheroma may undergo fatty degenerative changes.

In consequence of this increased bulk of blood constantly in the pulmonic circulation, there is a certain amount of interference with the respiration; the excess of blood within the thorax diminishes the space within which the lungs may expand on each respiratory effort. The breath is short on effort. As a result of high vascularity in an organ, we get a development of connective tissue, and this occurs in the lungs, going on in time to cirrhosis, or deposition of pigment, etc. In elderly persons we get emphysema, as the lung vesicles are torn up by respiratory efforts. In young subjects with normal tissues, emphysema is comparatively rare; in old persons, where the lungs are involved in the general

tissue degeneration which prevails, the lungs are readily torn, and emphysema set up. The violent efforts at respiration entailed by the diminished space for the lung to play in, lead surely in time to rupture of many air-vesicles into each other in advanced life. There is congestion of the bronchial mucous membrane with bronchorrhœa, which is often very troublesome.

It is well to be clear about this bronchorrhœa. It is not due to congestion of the pulmonic capillaries, for it does not show itself until the right heart has failed somewhat; and again it is relieved by the administration of digitalis, which, by its effects upon the right heart, raises the blood pressure in the pulmonic circulation. It is due to the congestion of the bronchial veins; and is a serous oozing from the venules, as is the effusion from the peritoneum, or the gastric catarrh found when the tricuspid valves leak. It is relieved by acting upon the right heart by digitalis; as is also the cough of congestion of the pulmonic circulation,—the "heart cough,"—with which all are familiar.

The pressure on the walls of the pulmonary vessels in mitral disease with a strong right ventricle may lead to hæmoptysis; or the blood may be poured into the lung structure, forming true pulmonary apoplexy; or the masses of blood may be localized, forming black clots, the size of a billiard ball, the infarctus Laennecii. "This form of apoplexy is very frequently found to be associated with active dilatation of the right side of the heart, and it seems to bear the same pathogenetic relation to the cardiac affection as cerebral apoplexy bears to active dilatation of the left side of the heart." (Rokitansky.) Pleuritic effusion and œdema of the lungs, more properly belong to the consequences of venous congestion from tricuspid failure, and will be considered in that division of the subject.

Sooner or later the right heart yields, either by the tricuspid ostium becoming so large as the muscular walls dilate that the valves become insufficient, or by secondary implication of the tricuspid valves rendering them no longer equal to the closure of the normal ostium, or by a combination of both. Then the venæ cavæ become distended, and the jugular veins are seen to pulsate. Some jugular pulsations may be seen before the tricuspid becomes insufficient. It is caused by the reflux of blood through the tri-

cuspid before its flaps are driven together on the ventricular contraction. All the branches of the venæ cavæ become distended, and especially is this the case with the portal circulation, which is not provided with valves. Splitting up as it does in the liver, the portal vein is partially protected by minute divisions in the liver, otherwise great disturbance would be occasioned, by the want of valves in it, on tricuspid regurgitation. But, in consequence of this subdivision in the liver, that viscus becomes gorged with blood in tricuspid regurgitation, and pulsates with the regurgitating current driven backwards by the hypertrophied right ventricle. Liver pulsation is a distinct symptom of diagnostic value, while the venous fulness of the liver leads to a development of connective-tissue corpuscles in the interlobular areolar tissue; first, there is increase of bulk in the liver, then cirrhosis or atrophy as the connective tissue contracts; the liver tissue becoming much firmer and harder than normal. The liver is easily deranged by an excess of food when so affected, and great care in diet is requisite for the proper performance of its functions. There is serous effusion from the gorged venules into the bile-passages attending it, so that Oppolzer has given to this condition the term "albumicholie," and he further states that in this condition there is congestion of the mucous lining of the bile ducts with jaundice, and that both these conditions are readily affected by a common cold. The disturbances and changes in the liver are much more marked in some persons with tricuspid failure than in others.

The spleen is implicated in this congestion of the portal circulation; it becomes enlarged from the development of connective tissue in it, is larger than normal, and of firmer consistence, resembling a beefsteak in appearance when cut open. We know, as yet, nothing of the symptoms of disturbance of the functions of the spleen.

The stomach and intestinal canal are also deranged, and the disturbances set up in them by tricuspid failure are distinct and pronounced. There is fulness of the venules of the stomach, and from that springs catarrh, with its pathognomonic indication, "sense of fulness," even when the stomach is empty. The feeling of being constantly "too full" is a very common outcome of ad-

vanced cardiac failure. The secretion of gastric juice is impaired, and it is diluted with a serous fluid. Consequently its digestive power is impaired. The catarrhal mucus is readily folded over any solid food, which so covered cannot be acted upon by the solvent juices, themselves impaired in power. Dyspepsia is the result of taking any but liquid food; and the condition of the stomach should ever be borne in mind in the treatment of cases where the tricuspid has begun to leak. In this condition of the mucous membrane gas is often disengaged very freely in many elderly persons, and the eructations are persistent, and often very loud. The patient's condition is very distressing, and the pressure on the diaphragm, and on the right ventricle, causes much disturbance of the respiration and dyspnœa, with very unpleasant palpitation. This altered mucous membrane also causes morbid sensations and cravings for highly-spiced or unsuitable food; and this may lead to differences of opinion betwixt patient and doctor.

The intestinal canal is involved, and may be disturbed in two directions. There may be irregular and defective action of the bowels from imperfect nutrition of the muscular fibre of the bowels, calling for warm aperient medicines (for here the aperients must be warm and contain carminatives as much as in the case of the menopause), enemata, or soap suppositories (Trousseau). Or the congestion of the venules of the intestinal canal may lead to diarrhœa, which often furnishes great relief, and should never be interfered with without good reason, nor until becoming unquestionably excessive. King Chambers says, that in conditions of gastric and intestinal catarrhs, the use of compound kino powder is indicated, and that under these circumstances, especially gastric catarrh, it rarely constipates. Alternations of constipation and of purging may occur. Bleeding piles, or hæmorrhoids, are frequent in the subjects of cardiac failure. Hæmorrhage from these piles often gives great relief, and should not be rashly checked. When no bleeding has occurred for some time a flow of blood from some other organ may take place, as hæmoptysis, for instance, and be followed by distinct relief.

The effects upon the kidneys are pronounced. From the venous congestion albuminuria is very frequent in the later stages of

cardiac failure; and the appearance of albumen in the urine is as ominous as is that of dropsy. As the arterial fulness lessens during the progress of the heart failure, the bulk of urine steadily falls; it becomes high colored and dense, and from the imperfect oxidation going on, lithates, usually pink, are found in abundance. There are usually to be found exudation casts and fibrin cylinders from the tubuli uriniferi. If there also be old-standing kidney disease there may be found old, small, contracted granular casts alongside the dark recent casts; or, according to Basham, casts in a state of fatty degeneration. The kidneys are found enlarged, swollen, injected with blood, and from the venous congestion a growth of young connective-tissue corpuscles is set up in them. When attending the Pathological Institute at Vienna, it was quite common to see these recent changes from heart failure alongside the evidences of old-standing renal mischief. The two were quite distinct. This subject will be treated more at length in the chapter on the Gouty Heart.

The genito-urinary system is disturbed by the venous congestion. There may be vesical catarrh, prostatic fulness, or urethral catarrh, or even hydrocele from congestion of the pampiniform plexus (Oppolzer).

In women there are catarrhal and menorrhagic troubles from congestion of the veins of the uterus and vagina. Oppolzer thought that profuse menses often accompanied the arterial fulness of aortic regurgitation, and regarded it as a symptom of some value in that form of cardiac disease. My own observations have not been numerous enough to enable me to corroborate this statement.

Dr. Angus Macdonald has recently published a work on the "Bearings of Chronic Disease of the Heart upon Pregnancy, Parturition, and Childbed," which is of the greatest interest; but unfortunately he does not discuss the questions of whether leucorrhœa is common in women with heart disease, or what is the effect upon the amount of the menstrual flux. So far as my impressions go, for I have no exact data on the subject, these outcomes of fulness of the pelvic veins are not so influenced by disease of the heart as might *a priori* have been expected.

Dropsy is a common outcome of tricuspid failure, it may occur

without tricuspid failure, and it may not be present as soon as a tricuspid murmur is heard; but it is never long absent after tricuspid leakage is confirmed. It may show itself and disappear under treatment, and remain away for a distinct period, as seen in the case given in the chapter on Treatment. It may come and go fitfully; or it may be uninfluenced by treatment. Its appearance is dreaded by all lay persons, and few medical men are not alarmed by its oncome. In cardiac dropsy the lower extremities are usually the first seat of the trouble. The legs and ankles are all right in the morning, but are swollen at bedtime; and the œdematous parts readily pit on pressure. This "pitting on pressure" is an ominous sign. So long as the swelling is resilient, and the pit soon disappears after the removal of the finger, matters are not so bad. But when the pit remains in the white sodden limb, like the pressure of the finger in dough, then the case is serious. Sometimes, when the patient is confined to bed, there may only be a little "puffing" about the ankle-joint or the arch of the foot; but if the patient becomes at all worse, the swelling increases. At other times the œdema reaches the knee, and stays there a long time, or even recedes for awhile naturally or under treatment. In other cases again the œdema mounts up to the groins, and the scrotum is distended with fluid; or the loose tissue of the penis is full of water. The arms are not so commonly affected; and in the cases I have seen where they were involved, there was in each a suspicion of pre-existing renal mischief.

Sometimes dropsy will come on quite suddenly, often giving great relief, by the abstraction of so much fluid from the gorged veins. When dropsy comes on suddenly and extensively, there is a better prospect of its removal than when it creeps on gradually and insidiously; and if it be removed by treatment, it may stay away for a long time. Especially is this the case where the dropsy is partly renal, partly cardiac. If the dropsy be entirely or partially removed, and yet returns again under treatment, the prognosis is exceedingly bad. The heavy sodden legs are a source of great discomfort to the patient. In some cases, the belly is swollen and distended with fluid; but general anasarca is not found with heart failure, as it is in nephritis. Effusion may

settle in the most dependent parts, over the scapulæ, back, loins, and buttocks in advanced cases. At times the dropsy disappears to some extent, and is accompanied by increased difficulty of breathing. Then œdema of the lungs must be suspected; and relief may be obtained on the reappearance of the dropsy. As the case advances, there is effusion into the serous sacs, the peritoneum, the pleura, the pericardium, the ventricles of the brain, and even the tunica vaginalis; and the intellect is clouded by carbonic acid poisoning. Occasionally the immediate cause of death is œdema glottidis.

During this period, the skin, stretched by the effusion into the subcutaneous areolar tissue, is liable to certain lesions. An erysipelatous blush may spread rapidly over the attenuated skin, and the skin seems as if it would die bodily, and yet pass away. This occurred in a case of failing gouty heart, where the prognosis seemed hopeless. Not only was there a general blush below the knees, but certain spots seemed as if they would sphacelate. Sir Joseph Fayrer advised that the limbs should be freely pricked with a needle; they wept copiously; the tension being thus somewhat taken off the skin, it recovered itself, and in twenty-four hours, there was scarcely a trace of what had so shortly before looked so very alarming. At other times, an erysipelatous inflammation may follow incisions or needle pricks, and be very troublesome. The skin may weep spontaneously from a form of eczema, sometimes furnishing much relief.

How far venous congestion affects the skin and renders it liable to disease therefrom may not yet be affirmed. Hebra teaches that ulcers are ordinarily connected with obstructed circulation; and I certainly have seen very obstinate ulceration in the legs of females with heart affections. In the dropsy of uncomplicated heart failure, the treatment is very unsatisfactory; but where there is also a renal element, and for this it does not by any means follow that there should be albumen in the urine, incisions, needle pricks, and Southey's drainage-tubes are very useful. Their action will be much assisted by sweating the patients and administering sharp hydragogue cathartics. In combined heart and kidney disease even extensive dropsy may yield to active treat-

ment and disappear for years, and that, too, when the heart is organically diseased.

Effusions from the serous surfaces are not common till the last stages are reached, except ascites in some cases of mitral stenosis. When the thoracic and abdominal cavities are extensively filled with effusion, respiration is only possible in a sitting posture, when the fluids fall away from the heart and diaphragm by their own gravity.

Orthopnœa, or inability to breathe in the horizontal posture, is found in the later stages of heart failure, and the patient may leave his bed and live out the remainder of his days in an arm-chair when it is very severe. It may occur earlier, especially during paroxysms of asthma (cardiac). It is certainly connected with interference with the descent of the diaphragm, and is occasioned by flatulence and fulness of the abdomen. In the horizontal posture the contents of the abdomen press upon the diaphragm equally with the other portions of the abdominal walls; in the erect or sitting posture they fall away from the diaphragm by their own weight. Dr. Lauder Brunton teaches that in the supine posture the diaphragm has to overcome the action of the abdominal muscles; when up, only lateral pressure upon them is called for. It is not suggested that the mechanical explanation here offered is the full and complete explanation of orthopnœa.

Cerebral symptoms, the result of venous congestion from tricuspid insufficiency, are found in all cases of heart disease. They form prominent symptoms in the later stages, while psychical disturbances are seen in all stages, early as well as late. There is an insufficiency of arterial blood, so that the brain tissues are imperfectly nourished. The effects of this are felt early, or during sleep, when the nutrition of the brain is carried on. (When awake the heightened vascularity is associated with its functional activity.) Consequently, the patient has unpleasant dreams, is frequently on the brink of a precipice, or is in battle. The writer's father died of heart disease; in his dreams he went through nearly all the battles of the American civil war. It is needless to say that such sleep is not very refreshing. In aortic disease, persistent disagreeable dreams are rarely absent, even in the early stages.

Then follow psychical disturbances in the waking state. The character often is markedly changed; the person of steady purpose becomes irresolute; caprice, vacillation, and irritability, blended with suspicion, if there be a renal element in the case, are the leading characteristics; the patient is whimsical, touchy, ready to take offence, so that quarrels with the attendants are very common, and the doctor has often to be a peacemaker, and even with him sometimes his relations with the patient are stretched to the utmost point of tension. Before the case has proceeded to this length the cerebral impairment is such as to unfit the patient for business matters; his calculations are imperfect, and error and want of success are the consequences. A business man once said to me he felt as if he were a book, in which a leaf were missing here and there; he could not tell their exact places, but he was conscious of an omission now and then. That missing leaf may turn out a very serious omission in a complex calculation. All energetic business activity is undesirable in several ways for persons with diseased hearts.

As the case progresses distinct physical signs show themselves. There is danger of venous effusion and serous apoplexy, or even more marked and active symptoms may be manifested. In one case of failing gouty heart, vivid hallucinations set in, the patient waking up from a dream with the subject of the dream projected into the waking consciousness with distressing vividness, and persisting for some time, in spite of all reasoning on his own part, and the intrinsic impossibility of the vision being a reality. The case terminated with acute brain symptoms, of which the psychical factor was a well-developed mania of suspicion. There is more or less swimming in the head, vertigo, singing in the ears, headache, sense of fulness, and sensations of unsteadiness. The discomfort of the day is exchanged for unpleasant dreams at night. There is no longer any true repose. The patient is wearied and exhausted, and the mind is affected. The most obstinate of persons may become wavering. This is admirably shown by George Eliot in *Middlemarch*, where she makes old Peter Featherstone vacillate betwixt his two wills. Indeed, the whole sketch is admirable, delineating with the utmost fidelity the deathbed scenes of a case of heart disease, where there is also more than a

suspicion of a renal element in the case. The recognition of mental alterations as direct outcomes of the physical condition in advanced heart disease ought to be more general than it is at present. Many tiffs, unpleasantnesses, and disagreements would be avoided, if we recognized more frequently the fact that the patient can no more help his mental state than he can be held responsible for the albuminous condition of his urine, or the dropsy in his legs.

The features become altered; the outlines are blurred; the lips are full, and of a crimson or bluish hue. Vascular twigs are distinct on the cheeks or nose, or there may be unnatural pallor and a pasty look. Œdema under the eyelids is indicative of renal complication, and is often found of a flitting character, one day seen, then not seen again for weeks, manifest after exertion or a bad night, persisting in ill health; most pronounced first thing in the morning, less notable during the day; fulness of the eyelids, with a certain pallor and dryness of the skin, is an indication which the observant physician knows well is most significant. Then the face may become cyanotic, especially after an attack of dyspnœa, and there may be a numbness felt in one or more limbs, all suggestive of advancing changes.

When such an advanced stage is reached as has been above described, sudden death is very common. The overtaxed heart may stop in diastole, unable longer to contract. The exertion of emptying the bowels, of a change of linen, is fraught with danger. Acute œdema of the lung may close the scene suddenly. At other times the case progresses until carbonic acid poisoning appears. The evidence of the accumulation of carbonic acid in the imperfectly depurated blood is drowsiness, gradually increasing. The patient may be roused by a question so far as to give an answer by great importunity, but the eyes quickly close, and the patient dozes off again with failing respiration, until the highly venous blood excites the respiratory centres in the medulla to more energetic discharges and more extensive respiratory movements; then the patient wakes up with looks of alarm, for that summons to breathe takes often the form of a hideous dream. Painful and disturbing as these dreams are, the inclination to sleep is irresistible, and the drowsy patient, worn out by his

efforts, as well as poisoned with the excess of carbonic acid in his blood, drops off again into the fitful sleep, only to waken again in a brief time in terror and alarm. There is only one consolation when this stage is reached, and that is, it cannot possibly last long; human endurance has its limits. Failure of the respiration, lung œdema, or choking of the bronchial tubes close the scene; while the crippled heart may still be struggling away—the cause of death, and yet not itself the first to die.

Such is the history of heart failure when the final stages are reached; but, fortunately, sudden death may step in at any stage and cut short the downward progress. Nor does every patient manifest all the terrible consequences of heart failure just described, very fortunately for them. It is in women that these fearful final stages are mostly reached, and some women manifest a catlike tenacity to life, and continue to live long after they have prayed to die. Where the heart is organically injured death from intercurrent disease is more frequent, and more easily produced than in perfectly healthy persons. If the heart mischief be of a stationary rather than a progressive character, as occurs in some forms of valvular disease, the muscular compensation may be sufficiently perfect for life to be maintained for years, if quiet can be maintained and the patient and the system generally can "level down" to the point of the crippled heart, and so a new equilibrium be attained. At other times the mischief is progressive and the direction of the case steadily downward; the extending disease in the valves goes on necessitating equally constant muscular changes to compensate the valve mischief, until the reparative powers of the system are worn out at a comparatively early period.

As to the subjects of chronic heart disease it is possible to sum up in the four following conclusions:

1. When heart disease exists with venous stagnation, many structural changes in the viscera follow, which pathological changes are accompanied by manifestations of functional disorder.

2. Though all functional disturbances may not be the direct results of the changes caused by heart failure, but have arisen from causes so-called accidental, still such disturbances are more

readily produced where chronic heart disease exists than in persons structurally sound.

3. In chronic heart disease the margin betwixt ordinary health and death is lessened, and consequently the limits within which disease may oscillate safely are diminished; the range within which the pendulum may swing is smaller.

4. Under such circumstances many trivial disorders, which in healthy persons may be left to themselves, must be promptly subjected to their appropriate treatment.

CHAPTER V.

HYPERTROPHY AND DILATATION—CHANGES IN THE FIBRILLÆ—CAUSES OF HYPERTROPHY AND DILATATION—MODE OF PRODUCTION—DIAGNOSIS OF EACH FORM—PROGNOSIS—IS HYPERTROPHY EVER DESTRUCTIVE?—MANAGEMENT OF HYPERTROPHY AND DILATATION.

THE most important matter connected with changes in the heart is a proper comprehension of the nature of those muscular modifications known as hypertrophy and dilatation. Without such comprehension all diagnosis is sterile, and all treatment merely empirical. On it rests the correct interpretation of each case of valvular disease, without it the prognosis has no sound basis. It is desirable then, that the causes and mode of production of these modifications be described with all the care possible, and with such accuracy as the present state of our knowledge permits. Hypertrophy and dilatation are not two processes, but the complemental halves of one process—as we shall see.

As to the histological changes themselves there is some dispute still. Once it was held that the new growth consisted of a development of spindle-shaped involuntary muscular fibres, a less perfect form of tissue, liable to fail soon and undergo degenerative changes, as it was noted that hypertrophied hearts were liable to degenerative change—an observation well based. It is now known that there is no such growth of lowly muscular fibre. Then there is "false hypertrophy," where there is a growth of connective tissue within the heart-walls, which often adds greatly to the heart's weight but not to its power. As Sir William Jenner has pointed out, there is no increase of power then with such growth, but a decrease of it. The well-known large heart at St. George's Hospital, weighing over forty ounces, is a heart of this kind. Then it has been disputed whether the increase in bulk of the muscular fibre in hypertrophy is brought about by enlargement in length and breadth of the already existing fibres, such as is seen in the development of the gravid uterus; or by a

true hyperplasia, a development of new fibres identical with the normal fibres. The older pathologists, as Rokitansky and Förster, held the former view, while more recent pathologists follow Rindfleisch in regarding it as a true hyperplasia.

Probably the growth of the heart-fibres is like that of voluntary muscular fibre. The biceps can be developed by voluntary effort and "the size of the individual fibres is unaltered, and the increase in thickness appears to be due to the development of new fibres, the elements of which previously existed." (McKendrick.) The tissue of a hypertrophied heart is darker in color and redder than that of the normal heart, and firm in its consistency so long as no degeneration has commenced. Whether there is corresponding development of the cardiac ganglia or not, is not yet ascertained; but it is probable. There is an experimental fact which throws much light on the development of muscular hypertrophy, viz., when a motor nerve is irritated, the bloodvessels of the muscle put in motion are dilated, and this, too, occurs even when the muscle is paralyzed by curari. What are the relations of trophic nerves to tissues and to motor nerves are not yet cleared up. But trophic nerves are connected with motor nerves; and functional activity of a muscle leads to its development, *i. e.*, hypertrophy, when the general powers of nutrition are good; to atrophy, when the nutritive powers are feeble; a fact which will help us much to comprehend how hypertrophy of the heart is set up in one person, while dilatation is the result in another—under the same set of circumstances.

Fig. 32.

Muscular Fibre of Heart. (From Rindfleisch.)

Increased demand upon the heart of a temporary character evokes palpitation; if permanent, hypertrophy is developed, provided the nutritive powers be good; if they are defective dilatation, either pure and simple, or blended with hypertrophy in every shade of proportion, is the consequential result. The cardiac chambers vary in size from time to time, and are not of absolutely fixed size. Nor are they always completely emptied on

systole. "The quantity ejected at each beat is governed more by the state of the rest of the body, than by the heart itself." (M. Foster.) Dilatation is a permanent condition of distension with imperfect emptying of the cardiac ventricle, and would always be the consequence of demand upon the heart if the arrangements by which hypertrophy is brought about were less perfect than they normally are. When the cavities of the heart are distended by a current driven into them with abnormal force, more rapid and more powerful ventricular contractions are set up, *i. e.*, a species of palpitation. We know that a heart can beat rhythmically when cut out of the body, and that the contractions are due to efferent discharges from the cardiac ganglia. Nevertheless the rhythmical action of these discharging centres is influenced by several factors, and increased distension of the cardiac cavities is such a factor, and a potent one. The sense of distension is received by the sensory nerves of the heart and carried to the cardiac ganglia, and more energetic and frequent discharges are given out. Though a heart will continue to contract rhythmically when cut out of the body, thereby proving that the measured ventricular contractions are excited by efferent discharges from the cardiac ganglia, still an effect is produced by the sense of distension of the interior surface of the heart. Both Ludwig and Von Bezold found that increased pressure within the heart accelerated cardiac action, when all nerve branches, both of vagus and sympathetic, were severed.

When this modification of normal processes is persistent, then hyperplasia of the muscular fibres follows from the dilatation of the bloodvessels, and the increased nutrition—of which the wider blood current admits. Such, broadly speaking, is the mode of production of hypertrophy. It may be set up, and in a well-nourished person usually is, before the distending process has proceeded so far as to be detected by the eye; at other times the new compensation is less perfect, and some dilatation goes on before the hypertrophic process can limit the dilating action—consequently the time permitted for the establishment of the new equilibrium is an important factor; while in a certain number of cases no new growth is attainable, and simple dilatation—elongation of the normal muscular fibres—results. Why hypertrophy should be attained in one case, and be absent in the other, is a matter on

which it is of cardinal importance we should have clear, well-defined ideas.

It used to be stated that hypertrophy was a modification of growth to enable the heart to overcome obstruction; a way of putting it which involves the idea of a "tissue-intelligence." It is really a growth of new fibres brought about reflexly in an intelligible manner.

The two sets of circumstances under which hypertrophy and dilatation, single or, much more commonly, combined, are developed, are these (1), obstruction to the onward flow of blood out of the ventricles; and (2) increased internal pressure from an abnormally powerful distending force. (Some other circumstances under which these changes occur, will be taken up after this section is discussed.) In obstruction there is an increase of internal pressure during the diastole as well as the systole, and the ventricle is imperfectly emptied on the ventricular contraction. "The first consequence of an obstacle to the circulation is, that the affected cavity is incompletely emptied of its blood. The gush of blood which enters the heart upon diastole, instead of finding the cavity empty, finds it almost as full at the beginning of the diastolic movement as it should be when that movement is complete. Blood continues to enter as long as the pressure of the afferent vessels upon their contents exceeds the power of resistance of the walls of the cardiac cavity." (Niemeyer.) A certain distension of the cardiac chambers is then the first consequence of an obstacle offered to the onward flow of the blood on the ventricular systole. It may be well at this point to review briefly the normal distending forces of the different cardiac chambers. The distending force for the right chambers of the heart is the blood welling in from the venæ cavæ. The propelling power of the blood in the veins is (1) the *vis a tergo* of the blood coming in from the capillaries; (2) the effects of the muscles of the limbs, which contract towards the trunk and compress the veins lying betwixt them and the elastic skin—regurgitation being prevented by the valves of the veins; and (3) the effects of inspiration and the atmospheric pressure on the extra-thoracic veins. The blood in the elastic veins wells into the right chambers of the heart until the internal pressure in the cardiac cavities and the veins is equal;

then comes the contraction of the muscular fibres of the venæ cavæ, continuous with the fibres of the heart, which contract towards the auricle and distend it; then the auricle contracts and actively fills the ventricle, which in its turn contracts and throws its contents into the pulmonary artery. The ventricle then becomes flaccid in diastole, so that it offers no obstruction to the blood welling into it, and thus the cardiac cycle is complete. The distending force for the left cardiac chambers is essentially the blood coming through the pulmonic capillaries under the pressure of the right ventricular systole. Probably the inspiration and the pressure of the elastic lungs on the pulmonary vessels are not without effect. But the chief distending force is the contraction of the right ventricle. The pulmonary capillaries are of unusual size, and the blood-flow through them is little broken, so that the contraction of the right ventricle is distinctly felt upon the internal surface of the left cardiac cavities. The blood comes in with a certain force from the pulmonary veins into the flaccid auricle and ventricle; the auricle is filled, it contracts towards the ventricle, and forcing so much more blood into it, raises the internal pressure on it, till the left ventricle contracts and expels its contents into the elastic aorta; whence the blood flows forward, its return on the aortic recoil being prevented by the valves at the base of the aortic column. There is a gradual gathering of distending force from the slow welling from the venæ cavæ into the right heart to the active distension of the left chambers by the current coming in from the pulmonary veins. The blood flows from the pulmonary veins with some distinct force, and the current runs into the left chambers until the pressure within the cavities and the veins is equal; then the auricular contraction adds to the pressure within the left ventricle, and the ventricular systole follows. That the distension has to do with the systole, though that essentially depends upon the rhythmic discharges of the cardiac ganglia, is proved by what is stated before at page 21; and also by the fact that in intermittency the ventricle does not respond to one auricular contraction, but that a second (or even more in some cases) auricular contraction is required to fill the ventricle, when the auricular systole is seen to run into and be followed by the ventricular systole. When then, from some ob-

struction offered to the outflow from the left ventricle, the chamber is imperfectly emptied, and is partially full when the diastole commences, there is the normal amount coming in from the pulmonary veins, and thus the ventricle is overdistended, and then rapid and energetic contractions follow.

If the muscular power is equal to the tax upon it, no excited action is set up. But if not quite equal to the tax, then excited contractions follow—that form of palpitation which is muscular, and which is the active evidence of inability. If this new process be maintained, then hypertrophy is brought about in the manner described above, viz,, by dilatation of the vessels of the coronary circulation. If the nutritive powers are good, and there is a sufficiency of time allowed for the formation of this new equilibrium betwixt the work to be done and the muscular power to do it, then there is pure hypertrophy without perceptible dilatation; if not, there is more or less dilatation with the hypertrophy—the more the hypertrophy and the less the dilatation, the better the resultant new equilibrium, and the greater the patient's general powers; the greater the dilatation and the less the hypertrophy, the worse and less perfect is the new balance attained. It will be well for the reader to grasp this thoroughly, as it is of the greatest value in directing the treatment of dilatation.

The effect of an obstacle offered to the blood-flow is, then, an imperfect ventricular systole, and the presence within the ventricle of a certain and abnormal amount of blood at the commencement of the diastole; and consequent thereupon an increase in the internal pressure upon the ventricle; a condition of temporary distension of the cardiac chambers which will result in hypertrophy or dilatation, according to (1) the amount of distending force; (2) the general nutritive powers; (3) the time allowed for the formation of the new equilibrium; and (4) the sex of the patient; subjects which will be spoken of more at length a little further on. The forms of obstruction which lead to these trophic changes are, aortic stenosis; pressure of a tumor or aneurism on the aorta; the distortion of the spine by rickets (humpbacks often die young from heart disease so caused); an atheromatous ring in the ascending aorta; and the changes which are caused by violent sustained efforts checking the blood flow in the arteries; and,

more than all others, arteriole contraction, interfering with the blood-flow out of the arteries. Of the first four causes little need be said—they are self-obvious. A few words upon the fifth cause may not be out of place. It has been little attended to except by Wardrop ("The Nature and Treatment of Diseases of the Heart"), who, under the title of "the musculo-cardiac function," describes the effects of the action of the muscles of the limbs upon the arteries. "When the muscles," he says, "contract within their sheath, they compress the arteries, and so check the blood-flow in them, and some cross the arteries to some extent." To exhibit this relative position of the arteries and muscles, we have only to examine a vertical section of the thigh about the middle. "The arteries are seen inclosed in their sheaths, and surrounded by the muscles in such a manner that, when the limb is moved, and consequently these muscles contracted, their pressure on the arteries must necessarily impede the passage of the blood." This matter is worth bearing in mind, and will be referred to again when speaking of the changes associated with aortic valvulitis, the result of strain upon the valves. It has been found that heart changes are, as the result of strain, commonly associated with excessive use of the arms. The prevalence of aortic valvulitis amidst a certain class of workers, namely, hammermen or strikers, that is, men who wield "the big hammer," as it is termed; colliers, who work in constrained positions; bargemen, who put nearly every muscle of their body into action, when pulling with their long sweeps; and its prevalence among soldiers who have to go through their drill in strained and abnormal attitudes, has attracted much attention of late from Clifford Allbutt, Myers (in his "Alexandra Prize Essay"), R. Farquharson, Arthur Davy, and others, as well as the writer. Hammermen are so liable to enlargement of the heart with aortic regurgitation following, that when in Leeds, where such disease is very common, the writer visited several works, in order to observe the condition of the "striker" during his labor. His exertions are most severe during the brief time that the iron on the anvil is sufficiently hot to be workable; and the muscles not immediately concerned in striking are fixed, in order that each blow may be given with greater force and precision. By this general muscular activity the flow in many arte-

ries is interfered with by muscular compression, and at the end of "the heat" the striker's heart is found bounding and contracting most vigorously. It has been temporarily distended, and is recovering its normal size during the brief interval of rest betwixt the heats. From observations made then and since, I am quite convinced that a stage of hypertrophy of the left ventricle precedes the aortic valvulitis. Where the muscular changes are imperfect, palpitation and inability to maintain his toil cause many a man to abandon striking, and so save him from valvular disease. It is where the muscular changes are well sustained that the striker is enabled to work on until the high blood pressure in his aorta, from the hypertrophied left ventricle and the obstructed blood flow in the arteries, causing violent closure of the aortic valves, sets up inflammation in them in time.

Arteriole contraction is a great cause of changes in the heart-walls. It may be temporary, as in hysteria (Eichwald), or it may be permanent, as it is in chronic Bright's disease. The changes wrought in the circulation by renal cirrhosis are as follows: arteriole contraction which obstructs the blood-flow out of the arteries; then the ventricle has more opposition to encounter on its systole, and it is imperfectly emptied; and then hypertrophic growth follows; the hypertrophied heart on the one hand and the contracted arterioles on the other, cause the blood pressure within the arteries to be persistently high; the coronary vessels are well filled and the nutrition of the heart is well maintained; and the hypertrophy is sustained for years. When, however, the hypertrophy fails (how this is brought about will be discussed at length in Chapter VIII), as fail at last it does, then a process of dilatation commences in the degenerate heart fibres, and they yield to the high internal pressure on them. Not only is dilatation then set up, but, in some cases where the nutritive powers are feeble, the hypertrophy from the first is not perfect, and there is a recognizable amount of dilatation present. This is commonly the case with women, and in them we find hypertrophy with dilatation, where hypertrophy uncomplicated is the rule with man; except in the cases of florid well-nourished women, where pure hypertrophy is found along with chronic renal disease.

Then hypertrophy of the right side of the heart is found when

there is any obstruction to the blood-flow in the pulmonic circulation, either from disease in the lungs, as interstitial pneumonia, chronic bronchitis with emphysema, or from the vessels being gorged as a consequence of mitral disease, and in rare cases by stenosis of the pulmonary orifice. Imperfect emptying of the ventricle on systole and high internal pressure on diastole is the cause of enlargement of the right side of the heart; there being always dilatation as well as hypertrophy when the right side of the heart is affected; in this respect differing from the left side of the heart, where pure hypertrophy is common. Changes in the size of the heart are also produced, when the heart is placed at a disadvantage and so empties itself incompletely on its systole. Thus in pleuritic effusion and in pericardial adhesion we find changes in the size of the heart. In left pleuritic effusion the heart is usually pushed over to the right side, and much less commonly further to the left by right side pleuritic effusion, and at first palpitation is present, because the heart so displaced is placed at a disadvantage; but after a time it becomes enlarged, and then it can perform its work without excited action. In pericardial adhesion, too, the heart may undergo several changes. In some cases the pericardial adhesion is most marked along the track of the coronary vessels (Rokitansky), and then of course the nutrition of the heart is impaired, and the organ soon undergoes degenerative changes. But usually the bands of adhesion are situated elsewhere, and do not interfere with the nutrition of the heart, but only impede its action. So trammelled the ventricles cannot contract completely, and then hypertrophy or dilatation, or both, are the results, according to the nutritive powers of the patient and the demands upon the heart. The strong motor discharges of the cardiac ganglia cause nutritive changes to follow. The heart may thus be affected by extra-thoracic causes of displacement of it, as ovarian dropsy, notably, or enlargement of the liver. The displacement effects the same increase of internal pressure from incomplete emptying as is the consequence of an obstruction to the blood current.

Then again there is another cause of cardiac hypertrophy than those just enumerated, viz., increased distending force.

This is seen in the hypertrophy of the left ventricle which follows aortic regurgitation, and which also is very commonly found

with mitral regurgitation. In aortic regurgitation the hypertrophy is often massive, and the heart is so enlarged that it has been termed the *cor taurinum*. Yet there is no corresponding obstruction to be overcome in aortic regurgitation. In aortic stenosis the obstruction to be overcome is distinct and palpable, and yet the resultant hypertrophy is moderate in amount. In aortic regurgitation the obstruction to be overcome is comparatively small, and yet the hypertrophy is massive. It is not any obstruction to be overcome which causes the massive hypertrophy, it is the regurgitant current which evokes it. The ventricle is no longer distended by the current normally flowing into it through the mitral ostium; there is a new incoming current of far greater distending power, viz., the regurgitant current, driven backwards on the aortic recoil through the imperfect valves. This incoming current produces great distension of the heart-walls, and then massive hypertrophy follows. The hypertrophy is in proportion to the force of the distending current. The term "over-compensation" has been applied by some writers to this massive hypertrophy, and certainly it is over-compensatory to the obstruction to be overcome. How far this massive hypertrophy is, in its turn, destructive to the arteries will be seen later on in this chapter; but it is not over-compensatory to the regurgitant stream, which possesses a wedgelike distending power, before which the heart fibres stretch, and dilatation is always found, however strong the mass of muscular fibre opposed to the new current. The great distension to which the left ventricle is subjected in aortic regurgitation is the provoking cause of the huge hypertrophy found therewith. If it were not for this growth of muscular fibre the left ventricle would early on become so dilated that it would be paralyzed in diastole, as it is commonly enough in the later stages, when the muscular fibre becomes degenerate, and can no longer successfully oppose the distending force.

"A considerable degree of dilatation of the left ventricle arises in cases of deficiency of the aortic valves, and a smaller degree in deficiency of the mitral," writes Niemeyer; and if any further proof were required to demonstrate that hypertrophy and dilata-

tion are related to increased internal pressure, it is furnished by the fact of the enlargement of the left ventricle in mitral insufficiency. Here there is no obstruction to overcome; indeed, there are two orifices, and not one, for the escape of the blood on the contraction of the left ventricle; there is the normal orifice for its escape, and, in addition to that, the leaky mitral valve. There is no obstruction to be overcome it is true, but there is an increased distending force to be withstood. The blood, which escapes backwards through the imperfect mitral valve, meets the current in the pulmonic circulation, driven forward by the powerful and enlarged right ventricle, the veins and the enlarged and thickened left auricle are gorged with blood; and then the left ventricle, when flaccid in diastole, is filled more violently than is the case normally, and so it becomes distended and enlarged. Its hypertrophy follows its distension; indeed, it is the least complicated illustration of hypertrophy being evolved to limit dilatation that we have. It is in proportion, too, to the distending force, for though the hypertrophy and dilatation are distinct, still they never attain the proportions found in aortic regurgitation.

In these forms of hypertrophy there is always dilatation. Where there is a distinct increase in the distending force, there is always evident dilatation with the hypertrophic growth. Here the fibres yield to the abnormal force, however massive the hypertrophic growth may be. The dilatation is the first step, and the hyperplasia the consequence. "Dilatation of the heart, when arising solely from an increase of the pressure of the blood within the cardiac cavities, as a rule, is soon followed by eccentric hypertrophy, the continuous and abnormally active contractions of the organ giving rise to a multiplication of its muscular fibres." (Niemeyer.)

This must be ever borne in mind that, with increase in the distending force, hypertrophy is always combined with dilatation of the cardiac chambers; in obstruction to be overcome, without any increase in the distending force, as in aortic stenosis, there is pure hypertrophy, usually without dilatation. That is, there may be hypertrophy without any dilatation where there is an obstruction to be overcome; but dilatation is never absent with an increase

in the distending force. In mitral stenosis the same changes go on behind the mitral valve as in insufficiency, and the pulmonic veins are gorged and the auricle is hypertrophied; but no change takes place in the left ventricle, because the incoming current is broken by the narrowed ostium.

Then there is a form of hypertrophy which is subsequent to a pre-existing dilatation; that is, there is a condition of permanent distension of the softened heart fibre, where hypertrophy, unattainable at the time, is developed ultimately. "When we come to study pericarditis, we shall learn that there is a form of hypertrophy of the heart which is purely the result of dilatation. The immediate effect of the infiltration of the cardiac wall which takes place in this disease is dilatation; very soon, however, this is followed by hypertrophy, although no fresh obstacle to the outflow from the heart has arisen meantime." This statement of Niemeyer's is pregnant with information as to the means to be adopted when we wish to artificially build up hypertrophy in an already dilated heart.

To pursue the circumstances under which hypertrophy occurs, we find that it is associated with nervous conditions. Older writers speak of nervous hypertrophy; thus, Hope says, "Every circumstance capable of increasing the action of the heart for a sufficient length of time—a period which must be very considerable—may be a cause of hypertrophy. Those circumstances may be (1) of a nervous or (2) of a mechanical nature. The former class comprises all moral affections, and all derangements of the nervous function that excite long-continued palpitation." This view is borne out by clinical observation, and such hypertrophy is not rarely found with nervous females of active habits who suffer much from palpitation. It is also found in chorea and Graves's disease, where also there is much palpitation. Trousseau described Graves's disease as a neurosis of the sympathetic, and Niemeyer thought the vasomotor nerves of the coronary circulation chiefly affected. In this form of palpitation the view here put forward of the *modus operandi* by which hypertrophy is brought about is strongly corroborated. The excited nerve discharges not only produce palpitation at the time, but, by the associated dilatation of the bloodvessels, hyperplasia of the muscular

structure is induced in time.* So long-continued palpitation of reflex origin may lead to some hypertrophy of the heart. Of the rarer causes of hypertrophy of the heart-walls it may be said that excessive eating and drinking is one cause. Niemeyer states such hypertrophy is found among travelling wine-sellers; and Oppolzer states that increased frequency and activity of the heart's contractions leads to hypertrophy in time, thus agreeing with the view put forward by Hope. Such a case occurred in the Pathological Institute at Vienna during the time I was watching the post-mortem work there, and especially all morbid conditions of the heart and kidneys. The man, who weighed some twenty stones, came into the hospital with right-side paralysis, and soon died with severe hæmorrhage from the mouth and nose. He was a comparatively young man, under thirty. A large clot was found in the left hemisphere, and there was a very large heart of textural soundness. There was no apparent cause for the hypertrophy except his habits; his kidneys presented no visible traces of disease.

Then it is just possible that the fibres of the heart may be the subject of lipoma. Flint relates a case where the heart attained unusual dimensions in a young man of twenty-three, who died with severe hæmorrhage from the mouth and nose. As a rule, however, hypertrophy of the heart arises from discoverable causes, and is brought about in an intelligible way.

Dilatation of the heart is the first step in the causation of hypertrophy; and under certain circumstances hypertrophy is never developed, or to a very slight extent, and the case goes on into confirmed dilatation. Here a new equilibrium is attained by a general "levelling down," and the permanent condition of dilatation is accompanied by a general enfeeblement, until a balance is once more struck betwixt the general condition and the crippled heart. Such a condition is more common with women than men; not as anything peculiar, but as a part of the general rule that states of general or local debility are more frequent with women than men. Probably this is due to the drains upon

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* In considering nervous hypertrophy of the heart, it will not do to overlook the possibility of general vasomotor disturbance raising the blood pressure in the arteries, and so favoring the production of hypertrophy.

women; not merely the catamenial flow, but the mucous discharges to which they are so liable during the intermenstrual intervals of the mensual cycle.

Inflammation of the muscular structure usually accompanies both endocarditis and pericarditis, and often some softening of the muscle remains after the inflammation of the serous surfaces has passed away. Consequently, the heart-walls yield to the normal distending forces without any obstruction to the blood-flow, and dilatation follows. Even here in time, as Niemeyer states, the dilatation is surrounded by hypertrophy if the nutritive powers of the patient improve. Probably in some cases there is a restoration of the heart to, or near to, the normal size.

Dilatation of the fibres of the heart with enlargement of the cardiac cavities often follows conditions of passing debility, and may under such circumstances be recovered from completely. Two such cases are quoted in the first edition of this work, and, as the patients are still alive, the cases may be referred to here.

J. T., an elderly and hardworking woman, waited day and night upon her husband for twenty days, while he died of acute tuberculosis subsequent to a sudden hæmoptysis. What little rest she got was in an arm-chair by his side. At the end of this time she was much exhausted, and her legs were swollen. A few days afterwards she consulted me, and presented all the physical signs of dilatation with a rapid, feeble, irregular pulse. Under treatment in a few weeks she lost all her symptoms and returned to work as a field hand. This was ten years ago, and she is a hale woman now.

T. A. at eighteen had engaged himself to a farmer for the summer of 1868, and overtasked his strength persistently. He came home quite unable to work, with a weak irregular pulse and a dilated heart. After three or four months of treatment by digitalis and iron, he lost all his symptoms, and returned to work as a first-class farm servant. From inquiries made after him in July, 1878, it seems, "he is an excellent farm servant and commands a very good wage; he never feels any weakness at the heart now excepting after he has had too much beer."

In another case of like origin, viz., persistent overwork, but of older standing, so good a result could not be attained, and a

condition of permanent dilatation with diminished capacity for labor remained.

Conditions of exhaustion alike of the cardiac ganglia and of the heart muscle are apt to be followed by a state of heart distension, remaining persistingly as dilatation. Such a condition may result from fevers, diarrhœa, dysentery, etc. Here there is a rapid, feeble, compressible pulse, the dilated heart merely pumping a little of the contained blood into the aorta, the ventricle never being anything like emptied. Here it is that digitalis produces that rapid improvement in the general condition, with lowering of the pulse rate, which is so remarkably illustrated in some cases. Slower and more complete ventricular contractions follow the action of this agent on the cardiac ganglia. In women such a state of heart is often produced by menorrhagia, with or without leucorrhœa. In many women, especially corpulent women, at the menopause, the heart becomes weak, dilated, and fluttering in its action, and any exertion apparently is impossible. This condition may remain permanently, but more often it is recovered from as "the change of life" gets over, and the woman enters on that calm existence which follows upon sexual involution. Some ladies have a weak dilated heart, who neither improve nor get worse for years; who can only maintain life under the most favorable circumstances, but so placed go on for a long time with a heart that would break down completely in a charwoman in a month. Often, no doubt, the general condition is dependent on the feeble heart, while in other cases, no doubt, the heart merely partakes of the general feebleness, and in these latter cases a more complex plan of treatment may be necessitated than in the cases where cardiac debility is at the root of the general state.

The gravest and most serious cause of dilatation of the cardiac chambers is fatty degeneration of their muscular fibrillæ. This usually occurs in time with hypertrophied hearts, when the nutrition of the heart fails—when the arteries have become atheromatous, and from the diminished aortic recoil the coronary vessels are insufficiently supplied with blood. As long as the nutrition of the heart-walls is maintained, there is no enlargement of the left ventricle, but when molecular decay has cut down the hypertrophy's arresting power, then the dilating process sets in—never

to be arrested. The growth of new muscular fibres which is set up by continued distension of the cardiac chambers is no longer attainable; and no other means can arrest the dilating process; the hypertrophied heart commences to dilate, and passes into its final stage of gradually increasing failure. Of this stage of failing hypertrophy Niemeyer, who evidently studied these heart changes most attentively, says: "Degeneration of the hypertrophied cardiac muscle is much accelerated if the patient's nutritive condition be allowed to deteriorate. One of the most common of the diseases of aged and decrepit people is an eccentric hypertrophy of the left side of the heart, caused by endarteritis deformans, which, when of long standing, gradually changes into dilatation by degeneration of its muscular substance." Such enlargement of a heart previously hypertrophied is of the gravest omen; it is a downward progress that can be little affected by treatment, except to some extent delaying its advance.

Failure of the right side of the heart by dilatation occurs in many cases of chronic bronchitis and emphysema, after a long course of years; or is the beginning of the end in many cases of old-standing mitral disease. When resulting from commencing degeneration of the muscular fibres, no repair can be hoped for.

As to the cases where hypertrophy is attainable, whether it will be instituted in a pure form, or will be blended with dilatation in various proportions, depends upon several matters, as mentioned in p. 108, which may now be considered. We have seen that this tissue growth is a reflex action, originating in a condition of persisting distension, leading to more active discharges from the cardiac ganglia, with the associated widening of the bloodvessels of the coronary circulation. But for this to be attained in its highest form several different factors must be present.

1. The general nutritive powers must be good, so that the blood is rich in nutritive material, and conveys to the tissues the material out of which new fibres may be developed. If the blood be defective in quality, then the hyperplasia will be imperfectly developed and a certain visible amount of dilatation will be present. When then a dilating process is going on in the heart, it is of the utmost practical importance to put the nutritive

powers of the patient into their best possible form, by attention to the diet and the digestive organs.

2. Time for compensation. If the time for the striking of a new balance be insufficient, then the highest form of compensation, as Traube terms it, is unattainable; a less perfect form of compensation, or hypertrophy with dilatation, may, however, be feasible. If the demand be very sudden, as in the case of the rupture of an aortic cusp, simple dilatation, the lowest form of compensation, may also be possible. The muscular fibres elongate; "what they lose as it were in force they gain in length," may be turned thus—"the longer they grow the feebler they become." If the nutritive powers be good, and the time sufficient, perfect compensation by hypertrophy is practicable; but for this a full sufficiency of time must be permitted. No matter how good the nutritive powers, if a new equilibrium is suddenly demanded, a less perfect form of compensation is all that can be achieved. Consequently all demands upon the heart must be reduced to a minimum in order that the nutritive powers may have the fullest opportunity afforded them for the production of the most favorable new balance. Perfect quiet is as necessary as good nutrition. (It is by watching the natural processes that we must be guided in our therapeutic measures when dealing with dilatation of the heart, and trying to build up hypertrophy.)

3. Sex has a great influence. The nutritive powers of most women are inferior to those of men, and consequently pure hypertrophy is comparatively rare with them. The practical outcome of this is, that when the normal balance of the circulation is disturbed in women, the greatest care should be taken to foster nature's efforts to attain the highest form of new equilibrium.

4. Amount of distending force ever exercises an influence; and if the distending force be increased beyond the normal point, then pure hypertrophy is out of question, and a certain amount of dilatation is always combined with it. Thus we see that in aortic regurgitation, even though the hypertrophy is most massive, there is a distinct amount of dilatation combined with it.

According to the presence or absence of these different factors, so will hypertrophy alone, or hypertrophy with dilatation, or simple dilatation, be the result of increased pressure within the cardiac cavities during diastole. Even where dilatation is established,

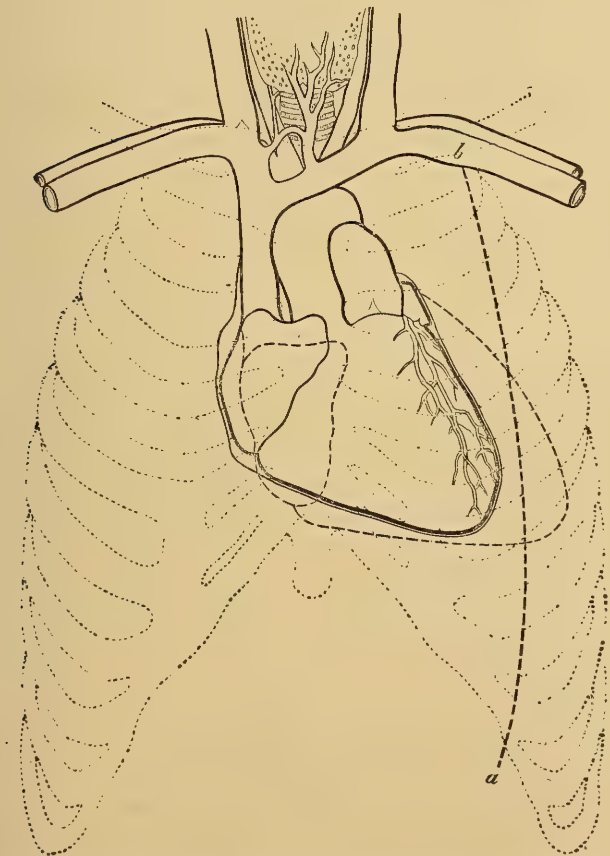
but has not existed long, I am inclined to think that a return to the normal size of the heart is possible; it being impossible to say that there is absolutely no hypertrophy, but certainly with no apparent hypertrophy. In proof of this may be adduced the two cases given above, to which others might be added (including cases of dilatation with a mitral regurgitant murmur, the murmur disappearing when the ventricle and ostium regained their normal size). This return to the normal size is certainly not in any way an *a priori* improbability, for we have good reason to know that the heart can become temporarily distended and recover itself; and this is especially true of the right ventricle.

As to the changes which go on in the heart in hypertrophy, there is an increase in the number of muscular fibrillæ, until a decided increase in weight is produced. The heart weighs normally, in the male, ten ounces, in the female, eight ounces; in hypertrophy it may weigh sixteen, or even thirty or more ounces; while in pseudo-hypertrophy a greater weight still has been reached. The walls of the left ventricle may become even an inch thick, and the right ventricle half an inch thick. Bizot calculated that hypertrophy was established when the left ventricle becomes six lines in thickness in the male, and five in the female; while two lines is the abnormal thickness for the auricles. As to the increase in volume, it may be general or it may be partial. We known that the fibres of the heart pass from the walls of one chamber to another; still the hypertrophy may be confined to one chamber or one side of the heart, according to the peculiarities of the case. The growth of the new fibres alters the appearance of the heart. When the left ventricle chiefly is enlarged, the heart is elongated, and the apex beat is lower than normal, and extends to the left; while when the right side rather is hypertrophied, the heart grows broader, and approaches rather a square than a triangular form.

Physical Signs of Hypertrophy—Inspection.—The apex beat will be seen to be lower and to the left of its normal place; it may be said that in left-side hypertrophy (the signs of which are now being reviewed) the apex is about an inch lower and to the left of the nipple line. Some bulging of the thoracic parietes over the heart is often seen in young subjects; it varies with the amount of hypertrophy. To palpation the heart's apex is felt as

a limited point driven forcibly against the thoracic walls at each ventricular systole. The apex beat is well-defined, powerful, and distinct, while there is a rounded heaving when hypertrophy is combined with dilatation. It may be felt down to the seventh or even eighth rib, and considerably to the left of the nipple—as far

FIG. 33.



Hypertrophy of the left ventricle. The continuous line marks the normal heart; the dotted line the increase. *a*, *b*, Linea mammillaris sinistra. (From Von Dusch.)

to the left of the nipple line as the normal apex beat is to the right of it. The impulse is forcible and heaving, but if dilatation also is present the apex beat is more diffused, and may even have a slapping character. Not only are the ventricles enlarged, but the left auricle may give positive evidence of its position, and

there is pulsation in the second or third intercostal spaces to the left of the sternum.

Percussion furnishes useful information in addition to that afforded by inspection and palpation. It tells that in hypertrophy the ordinary triangular space of complete cardiac dulness is enlarged. But in order to bring out the full value of the information afforded by percussion, it is necessary to bear well in mind the sources of fallacy, as an emphysematous lung covering the heart, and often rendering percussion of little or no value; while on the other hand, a patch of thickened lung, cancer, or tumor of the mediastinum, and pericardial effusion, will lend an apparent increase of size which is misleading. It is necessary, too, to avoid any mistake from an enlarged liver. Bearing the sources of error in mind, percussion will give useful information; it needs, however, to be done with great care, as it is not important to map out the area of complete dulness, but to ascertain with precision the area of comparative dulness—to be sure, so far as may be, of the outline of the heart. In computing this the sense of resistance conveyed to the finger is of value. In consequence of the interposition of the lung betwixt the chest-walls and the circumference of the heart, percussion requires the greatest care; but when such care is taken, it seems quite possible to realize the exact size of the heart, as any one who watched the late Professor Traube in the wards of La Charité, Berlin, must have felt. The dulness extends away to the left and lower than normal. The foregoing plate from Von Dusch, will give much aid in estimating the alteration of form produced by hypertrophy of the left side of the heart.

Auscultation gives the most decided information as to the size of the heart. On applying the stethoscope the first impression received is that of the heave of the cardiac apex. It is distinctly suggestive of power. Then the sounds are modified. The first sound is less clear than the normal sound, and partakes of the character of a "thud." The valve factor of the first sound is often obscured, especially at the base of the heart. The muscular sound predominates and the first sound is muffled and commonly prolonged. It contrasts with the clearer, louder sounds of hypertrophy with dilatation, where the valve sound is often loudly heard. In simple dilatation the first sound is often valvular

only, or nearly so. The first sound of hypertrophy is modified during an attack of palpitation. "During palpitation, the first sound sometimes becomes comparatively clear." (Walshe.)

An altered condition of the flaps of the mitral valve modifies the first sound and lessens the valve element. Then the second sound is commonly altered. Wherever the hypertrophy is connected with an increase of arterial tension—*i. e.*, in the great majority of cases—the aortic second sound is accentuated. It may resemble the crack of a whip, or the blow of the contact of two hard substances, or it may be clanging. It is most significant in elderly persons with calcified costal cartilages and emphysematous lungs (especially the anterior edges), where the first sound is lost at the base, and only faintly heard at the apex; here the loud aortic second sound is usually the best evidence of the condition of the hypertrophied heart—taken along with the firm incompressible radial pulse.

The modifications produced by the blending of hypertrophy with dilatation are as follows: The impulse is more diffused and extends over a larger area. It is not so strong and heaving as that of the pure form. The area of complete dulness is more square than the normal area; and the area of comparative dulness is extensive, and extends from an inch and a half from the right side of the sternum at the second costal cartilage, to the eighth interspace on the left side, and to three inches outside the nipple. Even a still larger area is occupied in some cases of aortic regurgitation. The lateral dulness is decidedly increased, and instead of the pointed apex of hypertrophy, we have an obtuse-angled triangle. On auscultation the heaving is found less pronounced and more diffused, and the cardiac sounds are clearer than in hypertrophy alone, and are loud and distinct; the valve sound of the closure of the auriculo-ventricular valves being very distinct, especially at the apex. The muscular element is not so preponderant, and the first sound is clear, and often sharp. The aortic second sound is loud and clear, but not so accentuated as in pure hypertrophy, because the blood pressure in the arteries is not so high. The action of the heart is somewhat irregular in many cases, and the disturbance of rhythm on exertion is usually marked. Palpitation is more frequent than in hypertrophy, and is readily provoked by effort. The pulse is

often full but wanting in firmness and only thin; not so sustained and more easily compressible than in pure hypertrophy.

The physical signs of dilatation are widely different from those of hypertrophy, an increase in the area of heart dulness being almost the only objective sign in common. On inspection there is a very faint impulse only discernible in very thin persons, where a diffused and feeble impulse may be found. In other persons it cannot be detected. It is usually below the normal position, that is below the fifth interspace. It is a feeble sound, slapping, and of an undulatory character. The rhythm is decidedly irregular, or becomes so on slight effort. The heart is broad and globular, and the area of comparative dulness is largely increased. It has been stated that percussion can distinguish betwixt a heart that is hypertrophied and one that is simply dilated, by the sense of resistance given; but this cannot be positively affirmed. On auscultation the first sound is short, clear, and abrupt, consisting almost entirely of the auriculo-ventricular valve sound. Its point of maximum intensity is below the normal point. The first sound often approaches the second sound in character. If there be some degeneration of the heart-walls accompanying the dilatation, a condition quite common, then the first sound may be very weak and feeble; while the second sound may be inaudible at the apex. There is something peculiar, yet scarcely describable by words, in the action of a dilated heart, but which once noted is ever after easily recognizable, and that is a break in the rhythm, a slight pause, with a sort of "roll over," and then the heart goes on again. This slight pause, which is not intermittency, but irregularity, is followed by a stronger contraction, which gives the impression of a diffused tumble of the heart against the chest-walls which is quite characteristic. It is rendered more frequent by effort; at other times, and this is when the heart is very much dilated and its walls very thin, the impression produced is that of a pulsatile sac, just expelling a little of its contained blood on each systole—a little off the top as it were. The rhythm is as follows:

Several beats of normal duration, then two or three short sharp beats, and a rather long diastolic pause followed by a longer, stronger systole; then the heart beats normally for a few beats, and then come the short strokes, the lull and the stronger systole, and so on. The variations of the rhythm may or may not occur at regular intervals; indeed all regularity may be lost. The more this irregularity is increased by slight effort the worse the aspect of the case.

Auricular Hypertrophy and Dilatation.—Under no circumstances do we find the auricles hypertrophied without dilatation. The opposite condition of dilatation without hypertrophy is not unknown in the right auricle, which has been found to consist of the layers of the endocardium and pericardium only, with the muscular fibres scattered and separate so that the auricular wall was partially transparent. These enlargements of the auricles are almost always associated with auriculo-ventricular disease. Thickening of the auricular walls is caused by the increased pressure upon them, whether in stenosis or regurgitation. Normally the auricles furnish no evidence of their existence, but when enlarged they both become apparent. In thin subjects the pulsations of the right auricle at the second right costo-sternal articulation are at times distinctly visible, and its enlargement is the source of much of the percussion dulness found under such circumstances to the right of the sternum. (Walshe.) It is well to distinguish it from pulsation due to aneurism of the ascending aorta. The left auricle can scarcely be said to have any physical signs connected with it ordinarily, as it lies behind the base of the heart and is deeply covered by lung; but when enlarged and hypertrophied its pulsations may become visible. To distinguish its movements from those of the ventricle, Dr. George Balfour places over each point of pulsation a bristle, carrying a flag, attached to the skin by a pellet of bees' wax. Dr. Sansom adopts the plan of attaching to each point a piece of adhesive plaster the size of a fourpenny piece. In the centre of each is a pin with the head to the skin, and the point outwards. To these pins spills of tissue-paper can be attached; the movements of these spills tell readily of the difference in time of the auricular and the ventricular pulsations, and are very instructive. The left auricle pulsates at the third and fourth left costal interspaces. (Sibson.)

Nature of change.	Inspection.	Palpation.	Percussion.	Auscultation.	Objective symptoms.	Subjective symptoms.
Hypertrophy.	Distinct powerful pulsation to left, and below normal left apex.	Limited heaving pulsation to left of nipple line, and down to seventh costal interspace.	Increase in dulness, slight laterally, but extending downwards and outwards.	Strong, dull, muscular first sound. Accentuated second sounds. No loss of rhythm.	Palpitation occurring while hypertrophy insufficient. Pulse strong and incompressible.	None. Compensation perfect.
Hypertrophy and dilatation.	More extended heaving, of greater width, and below normal points.	Impulse more diffused and less heaving, except in aortic regurgitation.	Increased general dulness; rather laterally than elongated, extending to eighth interspace.	First sound clear, valve element distinct. Often some loss of rhythm.	Palpitation on effort. Irregularity increased by exertion. Pulse full, but compressible.	Power limited. Feeling of dyspnœa on exertion. Effort affects the pulse rate distinctly.
Simple dilatation.	In thin subjects only can a diffused slap be seen, chiefly in the intercostal spaces.	Diffused slight pulsation rather than impulse below normal point.	Extended dulness, chiefly laterally, extending to seventh or eighth rib.	First sound thin and valvular. Second inaudible at apex. Marked loss of rhythm.	Palpitation common, even without effort, increased by effort with irregularity or even intermittency. Pulse small, weak, irregular, and compressible.	General condition of debility. Incapacity to make any effort without distress, dyspnœa, etc., following. Cardiac asthma at nights.
Hypertrophy and dilatation of right heart.	Distinct pulsation to right of sternum, from second to fifth costal interspace, not below normal point on the left side.	Impulse to right of normal seat, often powerful without corresponding change in character of radial pulse. Action may be more irregular than the radial pulse.	Dulness not to left, but to right, along the edge of the liver, and over the fourth, fifth, and sixth costal cartilages.	First sound strong. Accentuation of pulmonary second sounds. Impulse out of proportion to the radial pulse. Irregularity not felt so clearly in arteries.	Palpitation common, with dyspnœa, often irregularity. Both increased by exertion. Pulse small and compressible.* Fulness of veins of neck. Liver pulsation.	Condition of more or less feebleness. Attacks of dyspnœa common when hypertrophy is failing. The general indications given at Chapter IV sooner or later present themselves.

* Modified by coexistent states of left heart.

The physical signs of enlargement of the right side of the heart are of importance, and it is desirable to recognize them. The right side of the heart is always enlarged by a combination of hypertrophy and dilatation; it is never purely hypertrophied. It may become enlarged secondarily to disease of the left side, especially at the mitral orifice; or it may be primarily enlarged by disease of the lungs, the left side not being affected. It has a tendency to be enlarged in athletes, especially long-distance runners, gymnasts, swimmers, and divers. The efforts made obstruct the circulation through the lung, and then the right heart increases in power by muscular growth. As long as the growth keeps pace with the demand upon the right heart, the capacity waxes and the condition improves; or, to use their own parlance, they "train on." But when the efforts made are too severe, and in excess of the nutritive powers of the heart, then the compensatory growth is insufficient, and the power does not increase, but diminishes; they are then said to "train off." The questions of time for compensatory growth, general nutrition, and amount of demand, affect hypertrophy of the right heart, just as they do left-side hypertrophy. Sudden strain of the right heart from violent and prolonged effort is not at all uncommon; it is apt to be found in youths who overtax themselves in their athletic enthusiasm. When the efforts are not too great and too sudden, the right heart grows in size and in power; but the power to increase on demand is not alike in all persons.

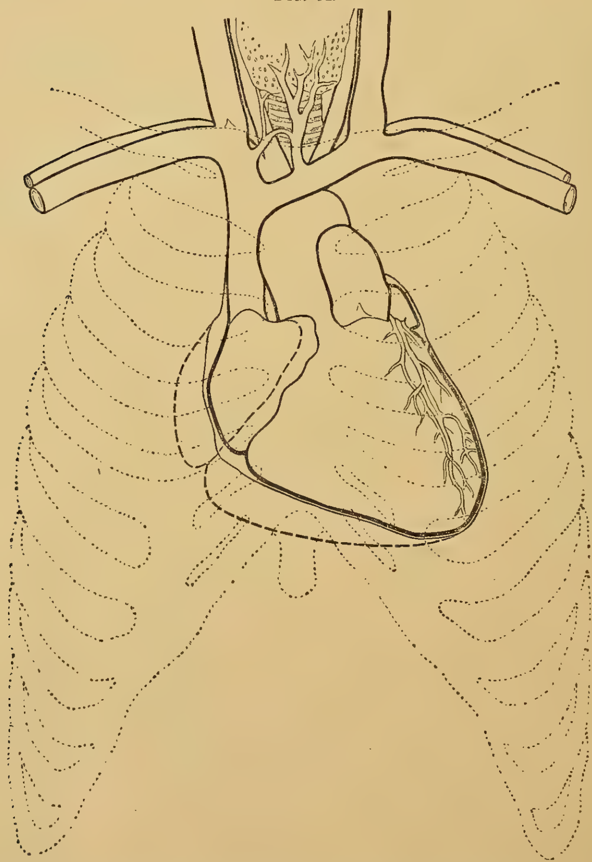
In the more slowly developed demand upon the right heart of mitral disease, or cirrhosis of the lung, or chronic bronchitis with emphysema, the growth in well-nourished persons is usually for long quite compensatory, and the case holds its own for years. In others the right heart grows but imperfectly, and the evidences of right-side failure are soon furnished, and the case moves swiftly downwards. Failure of the right heart is the common cause of death in acute disease of the lungs, and in cases of pulmonary congestion, usually intercurrent upon some chronic condition.

As to the changes of size and shape in the heart brought about by right-side enlargement, they differ from those of left-side enlargement. The apex beat is not altered, but the heart is enlarged laterally. It lies almost horizontally in the thorax, while instead

of the apex being decidedly the most dependent part, the right border forms almost a line from the apex to the fifth right costal cartilage nearly, but not quite, in a horizontal line.

The changes in size and position are well given in the following plate from Von Dusch's work.

FIG. 34.



Hypertrophy of right ventricle. The dotted outline shows the increase over the normal size, which is marked by the continuous line.

To inspection the beat of the heart is usually pretty distinct, diffused, and extending from the right apex towards, and even beyond, the ensiform cartilage. There is commonly no action

seen in the direction of the left apex, that is, provided the case be one of right-side enlargement uncomplicated by left-side changes. When there is both right and left side enlargement, then the signs are more complex. On palpation, a diffused heaving, distinct, superficial, and not without force, can be felt, extending from the right apex towards the fourth and fifth right costal cartilages. It is commonly accompanied by a distinct pulsation towards the second and third right costal cartilages, which is sometimes visible, caused by the action of the enlarged right auricle. To define the outline of right-side enlargement by percussion is a somewhat difficult matter. It is very difficult to define betwixt the dulness of the enlarged heart and liver dulness along the line of margin; and palpation and auscultation are safer guides for the beginner at least. Still a line of complete dulness may be traced along the right side of the heart, as seen in the plate. Then there is increased dulness, more observable on the right side of the sternum, commencing at the second right interspace, where the enlarged right auricle lies, and extending down to the fifth interspace. This increased area of dulness to the right of the sternum, and the absence of increased dulness to the left of the normal dulness, tells that the right side of the heart is enlarged.

On auscultation the first sound is heard distinct and strong, but giving an impression of superficiality, which separates it from the deepseated sound of an enlarged left ventricle. There is heard, too, at the ensiform cartilage, often very distinctly, the sound of the flapping together of the vela of the tricuspid valve. This valve sound is louder than the mitral valve sound heard at the left apex. Then there is also the accentuation of the pulmonary second sound, which Skoda regarded as almost pathognomonic of increase of size and power of the right ventricle. Then, in order to recognize that the signs of increased action in the heart are associated with right-side changes, it is important to contrast the cardiac signs with the radial pulse. The excited or powerful action of the heart is unaccompanied by any corresponding alteration in the radial pulse, as would be the case if the left side were the seat of the cardiac changes. There is not uncommonly excited action or palpitation of the hypertrophied chamber, or even irregularity in it, which is not traceable in the radial pulse. It is not unusual

to find in right-side enlargement a somewhat distinct irregularity in the heart on stethoscopic examination when there is no corresponding irregularity in the radial pulse; or at other times there is much more decided irregularity in the heart than is to be found in the arteries, as if the left ventricle only transmitted imperfectly the irregularity of the right heart. This is often very noticeable as the moribund condition comes on in thoracic disease.

Objective Signs.—The objective signs furnished by changes in the walls of the heart are significant of the form of change. Thus in pure hypertrophy, or perfect compensation, we have usually no evidences furnished of its existence, and extensive hypertrophy may be present, without an indication of any kind, so far as the patient is concerned. But some temporary trial, an increase in the cause of the change, as an arteriole spasm, or some passing cause of debility may reveal its presence, and send the patient to his medical adviser, who discovers hypertrophy. But it must always be borne in mind, that *in hypertrophy of the heart, palpitation is ever the evidence that the hypertrophy is insufficient—not that it is excessive*—except in purely nervous hypertrophy. This is a rule so important that it may be excused standing in italics; and the remembrance of it will often be useful. The immediate exciting cause may be temporary, but the palpitation indicates that for the time the heart is overtaxed, or temporarily incompetent. Palpitation is the outward visible sign of internal incompetence, and as such must it be regarded, whether hypertrophy is present or not (except in neurosal cases). The more pronounced evidences of cardiac failure—irregularity and intermittency—are not found with pure hypertrophy, unless it be at times of great general asthenia, or in some cases of aortic obstruction. The absence of objective signs, unless it be the powerful apex beat, characterizes true hypertrophy, and these signs when present, indicate that the hypertrophy is temporarily insufficient. But when hypertrophy is mixed with dilatation, then palpitation is readily induced by moderate effort, if at all sudden; though the heart's action may be regular and rhythmical enough in perfect quiet, or even in exercise or labor, if they be pursued quietly, steadily, and without fatigue. Palpitation, too, is readily produced by distension of the stomach or colon by gas, or other

accumulation in those viscera. Palpitation is also readily excited reflexly in the imperfect compensation of hypertrophy with dilatation. These objective phenomena are more readily excited when the condition is one of simple dilatation, where palpitation is set up by slight effort, and irregularity or even intermittency is mingled with it. This combination of unrhythmical action with palpitation on exertion is very significant; or there may be irregular or intermittent action constant, with palpitation readily superimposed by any effort; the unrhythmical action being more pronounced amidst the palpitation.

The simply dilated heart is ever unrhythmical in its action, and the want of rhythm is always rendered more pronounced by effort. The dilated heart has frequent halts, and beats of varying duration; often a number of comparatively steady, yet feeble beats, then a small cluster of very imperfect contractions, followed by a distinct pause and unusually strong contraction, and after that the usual comparatively normal beats are renewed. Sometimes the halt is not single but is repeated more than once, with its strong succeeding systole, before the heart resumes its quasi-normal beats. A gradual downward progression is often seen in old persons, when the heart is failing, and the irregular movements, at first only induced by effort, become permanently established; and any exertion is followed by further and more aggravated irregularity of action and prolonged halts; then the prolonged halt is found, even when at rest, with irregular action; and any exertion, however slight, produces—not palpitation, for the heart is no longer equal to it—but more distinct pauses, as the evidence of great enfeeblement. Along with this deepening of the objective signs, goes, hand in hand, a steady worsening of the subjective symptoms, with an aggravation of the subjective sensations.

Subjective Symptoms.—The subjective symptoms of pure hypertrophy are nil. When the hypertrophic compensation is perfect the patient is practically as good, that is as equal to exertion, as any ordinary person. But if the mixed form of compensation, hypertrophy with dilatation be present, then a comparative amount of vigor only is permitted, and the patient feels himself out of breath on any decided effort; and palpitation is easily induced,

not uncommonly accompanied by irregularity in the pulse. Consequently, effort becomes most useful for diagnostic and prognostic purposes. Effort by increasing the blood pressure within the heart may bring out a mitral regurgitant murmur from distension of the heart-wall, or an attack of palpitation, showing how readily the walls are overtaxed; though the sounds may be normal and the action perfectly rhythmic so long as quiet is maintained.

The bulk of urine passed is often most significant; and this is a matter too little attended to ordinarily. When the hypertrophy is good and the pressure in the arteries is high and well-sustained, then the bulk of urine is large and copious. When the hypertrophy is mixed with dilatation, then the bulk is less; where simple dilatation is present the bulk of urine is small; and it is of high specific gravity, and often is laden with lithates. The amount of urine—the measure of the blood pressure in the arteries, as Traube taught—is thus a distinct measure of the state of the heart; and the fall in bulk is ever suggestive that a hypertrophied heart is failing from degeneration of its walls. The amount of urine is an indication on which non-professional persons lay great stress; and from the bulk of urine passed the friends of a patient will often calculate the prognosis for themselves, especially in more advanced conditions of cardiac dropsy. Nor is the change in the bulk of urine a bad guide for the practitioner in estimating the value of his treatment.

The objective signs of right-side changes are also wanting so long as the muscular walls of the right chambers are well nourished, and quite compensatory to the new demand upon them. Of course there is the disease to which they are consequential to be taken into account. But when the tricuspid leaks, either from dilatation of the walls of the ventricle permitting enlargement of the tricuspid ostium, or from distortion of the tricuspid vela by valvulitis, or both, then the different signs, and with them the subjective symptoms, given in the preceding chapters, show themselves. There are jugular pulsations; a distinct fall in the bulk of urine, often with albuminuria from venous congestion; there is bronchorrhœa from fulness of the bronchial veins; there is œdema of the lungs, more or less flitting; a tendency to hæmoptysis; often cardiac dyspnœa from overdistension of the right

ventricle; faltering of the brain with oppression; and coldness of the extremities, with œdema. There may, or may not, be diarrhœa or ascites.

As to the liver pulsation which is found after the tricuspid leaks, and upon which the Germans lay much stress, it is due to the contraction of the right ventricle driving the blood backwards, so that the regurgitant blood causes the liver to pulsate from the filling of its veins. The liver is apt to become enlarged as a sequence of heart disease and its veins are distended, so that the regurgitant current, driven back by the ventricular systole, causes the whole mass to pulsate. It is probable that at times the liver pulsates before the tricuspid insufficiency is very marked, from the flow of the enlarged right ventricle being communicated to the liver through the thin diaphragm which separates them.

It is of great importance to estimate the relative amount of hypertrophy and dilatation in each case of right-side enlargement, as the aspect of the case varies accordingly. When the dilatation is unaccompanied by hyperplasia of the muscular walls, or fairly hypertrophied walls are yielding from tissue degeneration, then the breathlessness and panting are very distressing. As to enlargement of the right side, it carries with it an element of hopefulness; as upon its development rests so much of the future progress of the case in interstitial disease of the lung, chronic bronchitis, and mitral disease. Failure of the right side and the development of a tricuspid systolic whiff at the ensiform cartilage are of the worst omen, as indicating a failure which can never be compensated. When there is a new demand upon the right ventricle, and the system is unequal to the institution of good hypertrophy, the natural efforts may be aided by the means which are found useful in encouraging the development of hypertrophy in the left ventricle—when such growth becomes desirable. As to the concomitants of right-side failure, they are well put by Hope: "It must be recollected that in every organic disease of the heart, when palpitation becomes extremely violent and prolonged, both the impulse and the sounds may be diminished; in other words, the heart becomes gorged and incapable of adequately contracting on its contents, sometimes yielding a struggling convulsive impulse, with little sound and a feeble pulse,

and in an ulterior degree, especially during dissolution, scarcely producing either impulse, sound, or pulse. Suffocative dyspnœa, lividity, and extreme distress are always concomitant symptoms."

In the consideration of hypertrophic growth of the heart there are two very important matters still to be discussed. The one is the question, "*Is hypertrophy ever destructive?*" The other is "*The question of Permanency.*"

Can hypertrophy ever be regarded as itself destructive? is a question which must be answered in the affirmative. When it is developed in consequence of an obstruction at the aortic orifice, then it is purely conservative, and a more massive ventricle enables a normal bulk of blood to be driven through a narrowed orifice in a normal time. But when the obstruction lies in arteriole contraction, preventing the blood readily running out of the arteries, then the ventricular hypertrophy keeps up the high-blood pressure within the arteries, and this overdistension (Ueberspannung) leads to atheromatous changes in them in time. The hypertrophy which overcomes the obstruction ruins the arteries as a sequential result. In aortic regurgitation the hypertrophy which limits the dilatation produced by the unwonted distending force of the incoming current, driven backwards through the imperfect aortic valves, is ruinous to the arteries. The enormous ventricle at each systole drives with immense force an abnormally large mass of blood into the arteries, which are overdistended thereby. The recognition of this fact has led certain writers to speak of the massive hypertrophy of aortic regurgitation as "over-compensatory," because they did not clearly recognize the pathology of the condition. It is indeed "over-compensatory" as regards its destructive effects upon the elastic arteries. If we possessed any drug by which the heart could be made to contract more frequently and with a less bulk of blood expelled at each stroke in the hypertrophy of aortic regurgitation, we might do much to conserve an hypertrophy notoriously fleeting.

The question of the permanency of hypertrophy is one of great practical interest. It overlaps both prognosis and treatment. In calculating it, or attempting to artificially induce or conserve hypertrophy, the following points must be kept in view. Where-ever and whenever the supply of blood to the coronary vessels can

be well maintained, then the nutrition of the heart-walls can be kept up, almost, or comparatively, indefinitely. Loss of elasticity in the aorta by atheromatous change in its tunics, leads to imperfect nutrition of the heart-walls, and to fatty degeneration of the mucular fibrillæ. When the arteries are felt to be atheromatous, the prospect is clouded, but the end may still be far distant. In aortic stenosis the hypertrophy is usually durable and persistent. Also when hypertrophy of the left ventricle is found associated with mitral regurgitation, it is permanent and admits of length of days. But in aortic regurgitation, what betwixt the early deterioration of the arterial elasticity and thus diminished aortic recoil, and the escape of the blood backwards past the coronary orifices, the nutrition of the heart-walls soon becomes impaired, and the hypertrophy, though massive, is not durable; muscular degeneration soon leads to further and uncontrollable dilatation, ending commonly by cessation of the action of the ventricle in diastole.

Under favorable circumstances hypertrophy may be maintained for years, endowing the individual with capacity for exertion.

Its maintenance depends upon several factors, the first of which is the amount of work the heart has to do. Consequently, in those who must toil, the new equilibrium is sooner worn out than is the case where the patient can maintain a quiescent state persistingly. The affluent then have better prospects than the toilers. Then there is the factor of good general nutrition, where the poor are again at a disadvantage. Flint expresses the opinion that in all subjects of heart disease a moderate amount of exercise is good, and that the course is quickly downwards when exercise is no longer taken.

I do not think this statement open to the objection that the no longer taking exercise is the consequence of further failure of the heart, a construction that might be put upon it, but rather that a moderate amount of exercise is favorable to the maintenance of the general health and nutrition, on which so much depends. When we consider how purely mechanical the work of the heart is, we can understand that the best form of exercise is that which least tends to fatigue and exhaustion.

This leads to the subject of the treatment, or rather management, of conditions of pure hypertrophy. It is clear from what

has been said above that hypertrophy is not truly a disease to be combated, unless it be in the purely nervous forms; but rather a conservative process to be encouraged. Any attempt to remove it by the starvation plan of Albertini and Valsalva, is futile and inoperative; were it to produce any effect it would be to substitute the more serious condition of dilatation for the comparatively safe one of hypertrophy—to remove nature's check to the dilating process. Of course, in conditions of lithiasis such a plan would be efficient by removing the exciting cause of the increase in the tissue growth, and so permitting of a return of the heart to its normal size, as is asserted to be the case with the hypertrophy of pregnancy, which Larcher and Ducrest say passes away after parturition. Such doubtless is the explanation of the removal of hypertrophy by low diet and iodide of potassium in some cases; one of which was described to his class by the late Professor Elliotson, with such emotional excitement that he broke out into tears. The only treatment is that of conserving it as long as possible. When palpitation occurs with fairly good hypertrophy, digitalis should only be exhibited in small doses. Where the natural efforts are only equal to less perfectly compensatory growth, and dilatation is present, then larger doses may be given with advantage.

The great lesson to be learnt about hypertrophy of the heart, therapeutically, is the one of how to assist the natural efforts to attain it, when they are not quite sufficient of themselves. The principles to go upon are these. To reduce the demand upon the heart, or, in other words, to afford a longer time for the compensatory muscular growth to become established. Quietude, then, is the first matter. Then the general nutrition must be maintained by good food, by hæmatics, as iron, by tonics, and if necessary, cod-liver oil. Then the energy of the ventricular contraction can be increased by the administration of agents like digitalis, belladonna, and strychnia, which act upon the cardiac ganglia and excite more powerful efferent discharges from them. By these means muscular growth will be encouraged, and if pure hypertrophy cannot be attained the dilated walls may be enabled to develop more tissue growth, and dilatation be supplemented by hypertrophy, thus adding to the heart's power. Hypertrophy,

then, tends to maintain itself, while dilatation tends downwards. The less perfectly the arteries are filled the less perfect the tissue nutrition everywhere, including the heart itself. In attempting to aid nature's efforts to develop hypertrophy we must follow the natural indications ; we do not institute something new, we only further efforts which are started by the natural powers of the organism.

In that form of hypertrophy with dilatation found in elderly persons, where a heart once purely hypertrophied is commencing to dilate and yield as the consequence of mural decay by fatty degeneration, our measures can necessarily be but palliative ; all chance of curative measures has long since passed away. Here all our efforts, however earnest, can but delay the inevitable end ; still in some cases a fair amount of success may be attained, and life be prolonged.

In the treatment of conditions of dilatation of the heart many points have to be considered. The first, of course, is that of rest for the enfeebled organ. A few days in bed will often, without any medical treatment, work great improvement in a dilated heart. This is commonly seen in the heart cases taken into hospital. The heart which has been failing under the demand of toil, recovers itself in bed and gathers power, so much so indeed that many persons with feeble hearts are enabled by an occasional stay in hospital to work for a living during the intervals. So great is the effect of rest in such cases commonly that many men think no other treatment called for or of any utility. This, however, is a mistake, and the treatment of a dilated heart can be carried out on definite principles. The good food, iron, and digitalis, recommended for the treatment of hypertrophy and dilatation, are still more indicated in simple dilatation. The lines laid down in the Chapter on Treatment must be carefully followed out. All exhausting discharges are to be arrested, especially menorrhagic and leucorrhœal discharges in women. If there be diarrhœa present it must be treated. If the digestive organs are impaired, mineral acids with vegetable bitters are of service; if there be much gastric irritability then bismuth is of service. In the feeling of distension so pathognomonic of heart failure, and which appears to be due to fulness of the gastric venules, astrin-

gents are of little or no value; it can only be relieved by improving the circulation generally. In all chronic disease the condition of the stomach must always be the subject of solicitous care, and this is as true of cardiac dilatation as of phthisis even. The food must be nutritive and of the most easily assimilable character, given in small quantities at comparatively short intervals. All effort, as straining at stool, must be avoided. If necessary, the bowels must be regulated by some warm aperient; if there is diarrhœa present, which is clearly an oozing from the full venules of the intestinal canal, it must not be rashly interfered with. Often a certain amount of purgation, after the heart has been steadied by the exhibition of digitalis, is of great utility, getting rid of much of the watery constituents of the blood, and so improving its quality. Cod-liver oil may be given after the stomach has gained tone, with advantage, and aids in the development of new muscular tissue. As to the use of digitalis it must be pushed freely in much larger doses than are indicated under other circumstances; indeed it may be questioned how far toxic symptoms can be induced by anything like medicinal doses in conditions of advanced dilatation. It is often well to add to it strychnia and carbonate of ammonia, which also act on the cardiac ganglia, and, still more, act on the respiratory centre in the medulla, so that respiration is improved; a matter not unimportant in the embarrassment of the breathing which is so constant an accompaniment of cardiac dilatation. It must be clearly borne in mind, too, that the dose of digitalis which seems sufficient while the patient is quiet in bed, is not enough when the heart is taxed by the demand of labor. When the patient leaves the hospital the dose may have to be doubled, or trebled, before it is sufficient to keep the heart steady. This is a point too commonly overlooked, but is well worth remembrance. Then iron may be exhibited freely. Fuller writes: "In many instances I have known the physical signs of dilatation improve materially, and the general symptoms almost wholly disappear after a course of iron has been steadily persisted in for the space of three or four years." If desirable, arsenic may be added to the chalybeate, especially in the cachectic states. Belladonna may be given in those rare cases where digitalis disagrees, but in my experience

belladonna is more serviceable in neurosal and irritable conditions of the heart, than in actual failure of that organ.

Finally, the associations of dilatation must be clearly recognized. If of late date, and due to passing causes, it may be quite recovered from, as in the two cases given in this chapter; when of old standing in women in good circumstances it may be relieved, and the general condition much improved by careful treatment, though anything like cure is out of all question; when dilatation is due to fatty degeneration of a heart once well hypertrophied, then the prospect is gloomy indeed, and the end not far distant, however energetic and skilful the treatment may be.

CHAPTER VI.

ACUTE ENDOCARDITIS AND ITS SEQUELÆ.

Inflammation of the lining membrane of the heart is a common disease; serious rather from its consequences, or its associations, than in itself. It is found rarely as an idiopathic affection, and is most frequent as a concomitant of acute rheumatism, then of scarlatina, other exanthemata, Bright's disease, and typhoid fever. In a destructive or ulcerative form it is found with pyæmia and conditions of septic poisoning. It is also found as the result of the administration of lactic acid medicinally. (Balthazar Foster.)

The association of endocarditis with the presence of acid in the blood is curious. In acute rheumatism there is lactic acid, in lithiasis there is uric acid, and Virchow has found the blood to have an acid reaction in pyæmia.

Pathological Anatomy.—In the first stage there is injection-redness, when the endocardium is injected with points of twig-like vascularity. This inflammatory injection must not be confounded with the mere staining of the endocardium with the coloring matter of the blood, a post-mortem examination occurrence which not unfrequently happens, the endocardium being of a dull dark red, or almost crimson color. The inflammation is of the parenchymatous form, and young cells are developed in the subepithelial tissue, causing the membrane to lose its lustre, and to appear swollen. The serous membrane swells and is thickened, especially the free edges of the valves, and contains soft young cells in abundance. Among these cells are fine fibres and spindle-shaped cells. On the surface of the endocardium are found vascular growths, or villi, often in small aggregations, giving the surface a warty appearance.

These must not be confounded with fibrinous adhesions to the surface of the valves. They occur together, but must not be

confounded. These fibrinous deposits are liable to become detached by the blood current, by which they are borne away—to form emboli somewhere, not rarely in the left middle cerebral artery. The free edges of the valves become thickened and swollen, and the fibrinous deposits are most common where the free edges of the valves come in contact with each other—that is, the auricular surface of the mitral valves and the ventricular surfaces of the aortic cusps.

The endocardial inflammation is usually general over the left heart, though the mitral valves are most commonly the parts damaged thereby; and is accompanied by a certain amount of inflammation of the muscular tissue beneath the endocardium; and very frequently by pericarditis. The softening of the muscular tissue may lead to dilatation of the cardiac cavities, and if the softening be pronounced at some one spot, bulging, or true ventricular aneurism may follow. The musculi papillares are involved, not merely their investing endocardium, but their entire muscular structure. This may lead to their elongation, so that the mitral flaps may wash backwards on the ventricular systole; or to irregular action, and to insufficient closure of the valve in the comparatively early stages. The after-history of acute endocarditis will be taken up later on, and will be contrasted with sclerotic or contracting endocarditis. It is the proliferation of the connective tissue corpuscles in the fibrous structure of the valve flaps and their cordæ tendineæ, set up by the endocarditis, but continuing after the endocarditis itself has passed away, that is so serious, and pathologically so interesting.

Symptoms.—Endocarditis very commonly runs its course unnoted, and is frequently only discovered by physical examination of the chest. It is usually the mitral valve that is implicated; but in comparatively rare cases the aortic valve is acutely inflamed. Where there is no accompanying pericarditis it is doubtful if there be much pain. Sibson says the pain is sometimes, but not generally, severe. There is no tenderness on pressure unless there be also myocarditis. At first the heart's action is excited and the pulse is full, but this is only in the first stage. Afterwards the pulse becomes small, and the heart's action irregular. The sounds are altered by the thickening and swelling of

the free edges of the valves; and the valve elements of the first sound are wanting. When the muscle beneath the endocardium is much affected, the first sound is muffled, dull, and wanting in clearness. The first sound is usually further masked by a murmur at the apex, due to mitral regurgitation. Sibson says that the first sound is prolonged early on, and then merges into the systolic mitral murmur subsequently developed. There may also be a tricuspid murmur present. At other times there is an obstructive systolic aortic murmur when the aortic cusps are inflamed and roughened.

The second sounds are not usually affected. In acute aortic disease the sound is not so clear probably; while Sibson thinks the pulmonary second sound sometimes accentuated when there is mitral regurgitation. The production of a murmur is the characteristic indication of endocarditis.

In distinguishing betwixt endocarditis and pericarditis we are guided by the character of the sound produced. In endocarditis there is a murmur which has the same features as a corresponding murmur in chronic valvular changes, *i. e.*, the mitral murmur is conveyed to the left; the tricuspid murmur is heard at the ensiform cartilage; the aortic murmur is heard at the second right costo-sternal articulation. The friction-sound of pericarditis has no particular seat, it has its to-and-fro or triple sub-character. Walshe says he has never observed an acute obstructive mitral murmur.

Such are the physical signs. Pain is felt when there is myocarditis; and Piorry thought it was intermittent, or subject to rhythmic aggravation on each ventricular contraction.

As to any general symptoms, they are those of the concomitant affection of which the endocarditis is part. "The pulse is not remarkably accelerated, ranging between 80 and 120; the urine is simply febrile; cephalalgia exists more or less commonly, slight wandering may occur at night, but otherwise the head remains free; the respiration holds its natural ratio to the pulse so long as the orifices are not obstructed, and no secondary pneumonia has occurred; choreic symptoms are not induced if the disease remain simple." (Walshe.)

Where the case passes beyond the point usually reached, and

life is acutely imperilled, the symptoms deepen. There are evidences of obstructed circulation, either by the destruction of valves, the inflammation assuming an ulcerative character, or from the formation of clots in the heart. "Pulmonary obstructive murmur may be observed as the result of coagulation in the right cavities; but on the left side clotting of blood sometimes interferes with the production of a murmur." (Roberts.)

"The action of the heart becomes frequent, uneven, and irregular; the pulse, small, weak, irregular in force and rhythm, mounts to 130, 140, 160, or even more. Semi-syncope, pallor, coldness of surface, anxiety, and jactitation, inclination to orthopnœa (which the patient resists), from its increasing faintness, with, eventually, the symptoms of complete pulmonary obstruction, lividity of surface, turgescence of the face, prominence of the eyeballs, puffiness of the ankles, supervene; the brain suffers also congestively, as exhibited by fitful snatches of sleep, convulsions, delirium, and somnolence, lapsing into fatal coma. I have seen these symptoms in a minor degree and passingly in certain cases of endocarditis which terminated favorably, in all probability in those instances small concretions had formed, and subsequently undergone disintegration and solution. The symptoms of rupture of a chorda tendinea during the acute disease are extremely similar; the effects on the cardiac circulation must, indeed, be closely analogous." (Walshe.)

Prognosis.—Endocarditis, as ordinarily met with, is a disease which is serious to life from its consequences, rather than any danger in itself, except in the ulcerative form. The immediate danger is that of embolism. The vegetations which form on the inflamed surfaces of the valves get floated away in the blood current till they are arrested in some artery too small to permit of the further progress of the embolon, or plug. Here the embolon lodges, and cutting off the circulation, may lead to necrosis of the part whose blood supply is thus cut off. The most common situation in which an embolon is arrested is the spleen, then the kidney, and next the left middle cerebral artery. In spleen embolism there is pain over the spleen, tenderness, and swelling; when the embolon is lodged in the kidney, there is albuminuria and pain over the loins; when in the brain there is hemiplegia, usually with

aphasia. Rigors accompany embolism, and, when occurring in well-established endocarditis, are ominous; or a large clot may block up the femoral artery, or smaller ones lodge in the liver, in the coronary circulation, or elsewhere, as in the mesenteric artery; while very small ones may be found in the *arteria centralis retinæ.* These embolisms and their consequences form the chief danger to life in endocarditis.

Before proceeding to consider the treatment of ordinary endocarditis it may be well to consider ulcerative endocarditis, a very grave disease.

Ulcerative Endocarditis. — This is a form of disease found under certain conditions of blood poisoning. It occurs much more commonly in Germany than in England, and the following account is abridged from that of Von Dusch (*Lehrbuch von Herzkrankheiten*). This form of endocarditis commences with redness and injection of the tissue beneath the endocardium, which is swollen by a parenchymatous infiltration of connective tissue elements. There is also a free exudation from the surface, which is washed away by the blood stream. The epithelium is removed and the surface is dull, no longer smooth, but as if covered with a fine felt. From the connective tissue arise white gelatinous mucous patches, which appear on the surface of the valves as white granulations, having a grayish-red shade. These undergo a fatty degeneration with a dissolving of the connective tissue, and are washed away little by little in the blood current, forming superficial ulcers. These are accompanied by greater or smaller cell collections in the valves themselves, which force up the endocardium and perforate it in spots. In other cases there is fatty destruction of the connective tissue with formation of pus in fine small yellow points. These pass on to a molecular necrosis of the endocardium, with destruction of it and the formation of ulcers. This may proceed to ulcerative destruction and tearing of the valves. When this occurs at the base of a valve it may cut loose a portion of the valve, which floats freely in the blood current; a portion of a valve may so be severed from its attachments and be floated away *en masse.* The ulceration may involve the tendinous or muscular portions of the musculi papillares; or it may perforate the aortic valves, or open the two ventricles into each

other. In some cases destruction of a portion of a valve may lead to aneurism of the valves; at other times the ulceration extends into the muscular structure of the heart, and ventricular aneurism is formed.

Acute ulcerative endocarditis is seen either as a part of a general blood-poisoning with typhoid symptoms and adynamic fever, or under the cover of pyæmia, the heart symptoms being in the background. It is commonly accompanied by other acute affections, as myocarditis, pericarditis, pneumonia, or pleurisy, and by numerous so-called metastatic affections. In most cases there are initial rigors, with hot fits succeeding them, and at first these may have regular intervals daily (or even more frequently) resembling an intermittent fever, or the intervals may be irregular. Death may take place during the prolonged initial rigor. These rigors are succeeded by fever and perspirations which give no relief. In time the fever becomes more continuous, with slighter rigors. At the commencement there is vertigo, headache, great muscular weakness and prostration. The pulse mounts up to 130 or 150, sinking sometimes suddenly to 80 or 90, and becoming small and irregular. The temperature rises to 107°, the tongue is dry; then follow delirium, a drowsy condition and stupor. There may be sudamina, and often an almost roseolar exanthem may be observed. Sometimes this may be even papular or pustular, with more or less general ecchymosis of the skin. The appetite is lost at the commencement, there is vomiting and diarrhœa, with algor and cramps in the calves before death. In many instances there is jaundice, from the liver being implicated, either by metastatic abscess or inflammation of it. There is also enlargement of the spleen, with tenderness over it and the liver. The urine is scanty, concentrated, and often colored with bile, while it frequently contains albumen. The patient may have pain and oppression in the præcordia with marked dyspnœa, choking, or orthopnœa, or there may be few subjective symptoms. When pneumonia or pleurisy are present, there are pains in the side, cough with frothy blood-tinged expectoration, with the usual physical signs of these affections.

Among the objective symptoms is a systolic blowing murmur, or it may be diastolic. The heart's sounds may further be ob-

scured by exudation into the pericardium. On percussion there is generally increased heart dulness. Death commonly occurs from the adynamia passing into coma, or is sudden from the tearing away of the valves and chordæ tendineæ.

The prognosis is very unfavorable indeed, and the progress of the case to its termination is rapid. The prognosis depends partly on the general condition, partly on the endocardial changes. Whenever a systolic murmur is found to develop itself in the course of severe blood-poisoning or low septic fevers, then ulcerative endocarditis may be apprehended, and the prognosis becomes very bad indeed. The treatment has consisted of stimulants, ammonia, wine, camphor, musk, and the administration of quinine in large doses, in combination with opium; the subcutaneous administration of morphia, and the external use of ice and salt. But of course the treatment of such an affection is about as unsatisfactory as anything can be.

A chronic and localized form of ulceration of the endocardium is found at times where the pressure of a vegetation on the aortic flap of the mitral valve wears a hole in the aortic cusp pressed upon by it.

Treatment.—The treatment of acute endocarditis is a subject which will call out the physiological and pathological knowledge of the practitioner in each individual case. Here, indeed, we realize to the full the force of Latham's statement, "The treatment of diseases, rightly considered, is, in fact, part of their pathology." And as the Chapter on Treatment does not include acute conditions, the subject of the treatment of acute endocarditis will be considered here. As said before, endocarditis in itself does not usually imperil life—it is its consequences which we fear. The immediate treatment is that of the state with which it is associated. In acute rheumatism it is well to push the alkaline treatment actively. It is not found that salicylic acid exercises any beneficial influence over the cardiac complications of acute rheumatism. Opium may be given to relieve pain and to procure sleep. Hot poultices may be placed over the præcordium and relieve pain. The chest should be as little exposed as possible in examinations, the stethoscope being warmed before being applied. Cold applications over the chest are not much in vogue

in English practice. Bleeding, leeches, and blisters, are not indicated; and Niemeyer condemns energetically the plan of pushing mercury, which was advocated by Dr. John Taylor.

There are some points in the treatment of acute endocarditis which have forced themselves upon my notice since the first edition of this work appeared; and which seem to me worthy of being given in detail.

Pathological changes often give a direction to therapeutic measures; and our acquaintance with the morbid changes which are the sequelæ of acute endocarditis must guide us to its correct treatment. The inflammatory storm which passes over the endocardium lights up a growth of connective tissue corpuscles in the fibrous structure of the valves, and in the chordæ tendineæ, even to their muscular attachments. In consequence of the function of the valves and the chordæ tendineæ they can have no physiological rest when the seat of parenchymatous inflammation. This is a most serious matter. All are familiar with the enormous mass of callus which the late Mr. Hilton, in his admirable book on "Rest and Pain," describes as forming on the broken collar-bone of a washerwoman who did not give the injured part rest, but worked on. The growth of connective tissue corpuscles is lessened by rest, is increased by activity. The more the strain on the mitral valves and their attachments, the greater the development of connective tissue corpuscles in them after an acute attack of endocarditis. If these parts could be put at complete rest, the parenchymatous inflammation set up by the endocarditis would probably soon cease, too, and no permanent injury be produced. But then the parts cannot be put at complete rest; and, consequently, the growth of pathological connective tissue too commonly goes on till serious mischief is unavoidable. It is this growth and its subsequent contraction, which distorts and mutilates the valves, which we fear. If the growth could be arrested the subsequent injury could be limited. We can exercise no beneficial effect over the shrivelled and injured valves when that stage is reached; but we can, if we bear in mind the lessons taught us by pathology, exercise a profound influence over the early changes on which the later ones depend. We must remember the functions of the valves and their tendinous cords. Every

time the left ventricle contracts the flaps of the mitral valve are exposed to as much strain as equals the distension of the elastic arterial system. The more the contracting power of the ventricle and the resistance it has to overcome, the greater the strain on the mitral valves with their chordæ tendineæ, and the less their physiological rest; consequently the greater the growth of connective tissue corpuscles in the fibrous structure of flaps and cords. The true line of treatment is obviously and self-evidently to lower to the minimum the functional activity of the flaps and cords. In order to attain this the patient should be kept perfectly quiet in bed, for several days at least, after the inflammatory storm of endocarditis has passed away; until, indeed, we may fairly suppose the consequential parenchymatous inflammation set up in the tissues beneath to have also been brought to a standstill. By such means we will unquestionably limit the growth of connective tissue corpuscles, and with that the ultimate distortion we dread. Of course it is impossible to prove in such case the good we have succeeded in doing, or the evil we may have avoided or averted; but the principle is too clear to need any array of statistics, any host of collective cases to prove its claim to our confidence. The few cases of acute endocarditis which have come under my care recently have been treated on this plan with satisfactory results; and my friend, Dr. Hughlings Jackson, has adopted it with his cases at the London Hospital.

It is sad to think how, with the pathological facts of acute endocarditis and its sequelæ before them, authority after authority has gone on basing their claim to our professional confidence on the short time—the few days—which elapse before their patients can sit up or be discharged from the hospital. Surely this is the most pernicious thing that could happen—the thing, indeed, most conducive to future trouble. If they deliberately designed to prejudice the ultimate interests of their patients, they could have selected no line of treatment containing so many potential elements of success as the one they have chosen. It is true Dr. Sibson has observed the good effects of rest in the treatment of endocarditis, for he writes: "The whole chain of evidence points, then, irresistibly to the conclusion that the extent, severity, and permanent ill effects of endocarditis were much greater in the series of cases

that were not rigidly treated by rest, than in the series that were so treated." It is sad to think of the human suffering undergone by those who were not treated by rest rigidly enforced; such evidence was scarcely necessary to illustrate a principle so plain and self-obvious. But even in Dr. Sibson's elaborate work on endocarditis ("System of Medicine," volume iv) I fail to find any evidence that the plan of absolute rest was continued beyond the time of the acute symptoms; though, in speaking of pericarditis, he says: "Rest and support of the affected joint should be strictly maintained for several days after the disappearance of the local inflammation." And what is so necessary for joints is equally desirable for the valves of the heart; and rest, so far as it is attainable, should be afforded to them by keeping the patients supine in bed, and by keeping the blood pressure low, as by the administration of chloral hydrate. What is the importance or annoyance of a few days in bed compared to the terrible results of marked mitral valvulitis? Here, indeed, the immediate should be subordinated to the permanent treatment of disease. The immediate necessities of the patient may seem to call urgently for stimulants and digitalis; but with the remote consequences before our eyes these should only be administered to the extent that will stave off death from exhaustion, collapse, or syncope, but no more than will just achieve the end.

General quietude for weeks after an attack of acute endocarditis is indicated, as the cell-growth in the valves may not be quite over in a less time.

The Sequelæ of Endocarditis.—This division of the subject is one of the highest importance. And in order to give further force to what has just been said of the treatment of acute endocarditis, it may be well to consider the after conditions in various subjects.

And first it is desirable to bear in mind that the valve changes set up by acute endocarditis differ from those of sclerotic or contracting valvulitis, in that, in many instances, they can be brought to a standstill. A certain injury to the valves has been done, after which the condition becomes strictly static. If this stationary condition can be secured, the patient is little injured, and the muscular growth requisite for a new equilibrium is easily attained and further maintained.

The late Peter Mere Latham in his works devotes a whole chapter to "Consequences to life and health from the permanent unsoundness of the heart remaining after endocarditis.—1. Cases in which besides the permanent endocardial murmur there is no other symptom referable to the heart; 2. Cases in which besides the murmur there is occasional palpitation; 3. Cases in which besides the murmur there is constant palpitation."

Had the writer been familiar with Dr. Latham's writings some years ago, he would have been spared much labor in finding out for himself what Dr. Latham's works could have taught him. An extended observation of mitral disease furnishes cases where if the patient remain quiet there is no evidence of cardiac mischief beyond the mitral murmur. Such persons are seventy-five, or even more, per cent. of their former selves, and with quietude may reach a normal length of days. On climbing a hill, or running upstairs, they are made conscious that they are not quite what they were, but so long as they are quiet they suffer nothing. They are lowered down to the point of a quiet existence; in other words, "levelled down" to the position of their crippled heart, but no further. In a few happy cases even the murmur has disappeared under treatment, and the patient is apparently as well as ever he was. This is well shown in a boy, Fred Quire, a patient at the West London Hospital. He had a distinct mitral regurgitant murmur after rheumatic fever in 1874. Up to 1876 the murmur could still be detected; since then it has disappeared. The boy has grown very fairly, is a sturdy fellow, and can swim across the Thames and back again without any difficulty.

Then others are a little further injured; the murmur persists, and there is accentuation of the pulmonary second sound, indicating fulness of the pulmonic circulation. Such persons are easily put out of breath on any exertion; are liable to recurrent attacks of pulmonary congestion with hæmorrhage; but nevertheless are capable of living for years, even when a certain amount of toil is unavoidable (case, p. 58).

"Now when the heart bears the injury to its endocardium thus patiently for years, it is fair to infer that the injury is either so small or so fortunately placed as to offer no sensible obstruction

to the passage of the blood. A little ridge on the surface of a valve, a little granule on its free edge, or a little thickening or shortening of a tendinous cord, may be conceived capable of occasioning eddies and vibrations of the blood which can be heard, without producing any hindrance of the current which can be felt." Of all those who suffer rheumatic endocarditis and recover, but with some permanent detriment done to a portion of the endocardium, a large proportion, I suspect, fall under the foregoing description.

"There is yet another description of cases, including those who have a permanent murmur derived from acute rheumatism, and together with it a permanent palpitation. After the subsidence of the rheumatic attack, these patients may recover the aspect and many of the feelings and functions of health; but their heart is *always* found to beat with somewhat more than its natural force, and with very much more upon any considerable exertion. Now, from this palpitation, conjoined *from the first* with the persistent murmur, I do not know that we can justly infer that the heart has sustained *from the first* a detriment to its structure beyond the injured endocardium. But in such cases we are apt to think that we shall not have long to wait for the authentic signs of its more extensive disorganization, and we are apt to look for an earlier incapacity and an earlier death. Yet this need not be. Even here the changes for the worse are often very slow. Life, useful and enjoyable life, may endure for years, even for twenty-two years, as the following case will show, and then afford the prospect of enduring still." (Latham.) This case was a lady of thirty-six, who had her first attack of rheumatic fever at fourteen. At eighteen she had a second attack, "but the habitual symptoms referable to the heart were neither aggravated at the time nor rendered permanently worse afterwards. Ten years ago she had a third attack, which, for the time, brought upon the heart an extraordinary amount of suffering, but left it no worse than it was before. I considered that the mitral valve was unsound, and that the left ventricle was in a state of hypertrophy and dilatation, and the aortic valve not altogether uninjured."

Of course the life led and the occupation of the individual will exercise a profound influence over the progress of a case of

mitral valvulitis. If the patient must toil, then more strain is thrown upon the mitral vela, and the development of connective tissue in them will be encouraged and fostered. The question might be raised, speculatively, how far this growth in the structure of the valves and their chordæ tendineæ is not an attempt to give the parts physiological rest by checking all movement,—either soldering the valves down to the musculi papillares, or fusing the valve flaps together and so putting them at rest. But the conservative growth—if conservative it be—has destructive consequences of its own. General quiet certainly is indicated in all cases,—alike those that are static and those that are progressive.

Where the condition is static, and the mitral flaps are very nearly competent to close the ostium, the compensatory muscular growth in the right heart is readily achieved, and can be maintained for years, so that the patient is little below the line of normal health. But where it is progressive the same consequences are entailed as occur in the sclerotic or contracting form of mitral valvulitis, viz., a gradual narrowing of the ostium, or increase in the regurgitating current from the shrinking and contraction of the valve flaps, necessitating equally persistent changes in the muscular compensatory growth. Where these constant changes to achieve a new equilibrium are so necessitated, the case grows rapidly worse, because the nutritive changes required to maintain the new equilibrium become insufficient; the system is unequal to the constant demand upon it; and then the later stages of heart failure, with all its consequential results, follow.

The question might be raised how far the treatment that we must adopt to encourage the natural attempts to attain a new equilibrium by compensatory muscular growth, *i. e.*, the administration of digitalis, iron, and good food, does not aggravate the changes in the valves by throwing more strain upon them; but this only points the more clearly the paramount importance of limiting the first stage of the pathological process, viz., the development of connective tissue corpuscles in the structure of the valves—set up by the inflammatory storm which passes over the endocardium in acute rheumatism.

As to the effects of subsequent attacks of acute rheumatism upon

valves already injured by a first attack, opinions differ. In Dr. Latham's case, just given, no further injury was inflicted by the second and third attacks; while Dr. Sibson speaks of "the influence of previous valvular disease in rendering endocarditis more frequent and severe in cases of acute rheumatism." As to my own experience, many cases are known to me where a second attack of rheumatic fever has done no further injury to valves mutilated by a previous attack. For instance, M. D. had rheumatic fever when thirty-two years of age, for which she was attended by my father in 1843. This left her with mitral regurgitation of severe character. In 1866 I attended her for a second attack of rheumatic fever. There was no further heart complication. In 1878 I hear that her general health is much better than it formerly was, and that she now seldom suffers from palpitation.

When the acute endocarditis involves the aortic valves the case probably seldom remains static, but progresses steadily onwards, even if slowly. Aortic regurgitation is never stationary for long, and the cases I have seen of it, taking their origin in acute endocarditis, nearly all in girls, have advanced to their later stages in from four to eight years.

The great fact about acute endocarditis is to make our knowledge of its pathology direct our treatment of it, and to subordinate the immediate to the permanent interests of the patient, and to bear in mind "that the evil consequences to life and health arising out of the heart's permanent unsoundness left by endocarditis are often either stationary, at a small amount, for years, or very slow to advance and accumulate. If a reason for the fact be asked, it will be found in the stationary and unincreasing nature of the original endocardial unsoundness." (Latham.)

CHAPTER VII.

VALVULITIS.

By valvulitis is meant that chronic parenchymatous inflammation of the valves of the heart which constitute a very common and serious form of heart disease. There is a slow proliferation of connective tissue corpuscles, which in time distorts and cripples the valves and limits their functional activity. This growth may arise (1) from an inflammatory storm passing over the endocardium, and starting this cell proliferation in the valves; or (2) it may set in insidiously, and only be recognized when a certain definite amount of progress has been reached. The first form is commonly seen in the disease of the mitral valve which results from acute rheumatism or scarlet fever, and less frequently from other acute maladies of childhood. Here the acute inflammation of the endocardium is accompanied by a proliferation of connective tissue corpuscles in the structure of the valvular flaps, which commonly extends to the tendinous cords of the musculi papillares. In a few months after an attack of acute rheumatism, accompanied by endocardial inflammation, a mitral murmur of permanent character may be heard, telling of valvular change. In these cases very commonly a certain amount of distortion and mutilation of the vela of the mitral valve has been worked; but there the inflammatory process stops, and makes no further progress.

In such cases the prognosis of the case as to life is fairly good, and if the nutritive powers be well sustained a certain amount of capacity to undergo exertion remains. It is in dispute still as to what is the effect of another attack of rheumatism with endocardial complication upon valvular flaps once modified by a previous attack of inflammation. As a rule, serous surfaces once inflamed do not readily take on acute inflammation again; but opinions differ as to the effects of a second attack of acute endo-

carditis accompanying rheumatic fever. The diagnosis is difficult from the existence of the old murmur, and as patients rarely die at this stage, it is impossible to estimate the amount of the new injury except by the resultant consequences, which may not reveal themselves for some time after the attack. All that may be affirmed is, that in some cases further injury does seem to have been inflicted by the recurring attacks of rheumatic fever, with endocardial complications, while in others no such consequences can be detected. In other cases the chronic inflammatory process persists until such valvular ruin is worked that it is incompatible with the further existence of the patient; it may be not many months after the primary attack. The mitral disease of children and of adults is commonly occasioned in the manner just described.

In advanced life the origin of mitral valvulitis is less acute, and there exists a slow, stealthy production of connective tissue of a very chronic character. This is frequently accompanied by changes in the walls of the left ventricle, which may be due to the valvular lesion in mitral regurgitation, as we shall see at page 173, or may have other causal relationships. Of these changes overstrain is undoubtedly a, if not the chief, factor. The mitral valve has to sustain at every contraction of the muscular left ventricle a force equal to the distension of the arterial system. The aortic valves have only to sustain the force of the recoil, yet we recognize strain as the cause of the aortic valvulitis, found so commonly with the high arterial tension of chronic Bright's disease. Recent observation and experience incline me to hold the view that chronic mitral disease is also commonly found with the vascular changes, including hypertrophy of the left ventricle, which we shall see in a subsequent chapter are the results of chronic renal changes. The high arterial tension leads to ventricular hypertrophy; and the more powerful muscular wall, with its more energetic contraction, puts so much more strain upon the mitral vela at each systole. The flaps of the mitral valve are more violently closed when the blood pressure within the arteries is high, and so nutritive changes are set up in them in the form of chronic valvulitis. The occurrence of mitral disease, especially in old persons, and in the form of regurgitation, along with

an hypertrophied left ventricle and corded arteries, is too frequent not to suggest the impression that strain is a cause of mitral as well as of aortic valvulitis. In other cases the causation is different. In the present state of our knowledge all we can safely say is that in some persons changes in the mitral valve are found which are not associated with an acute primary attack, nor yet apparently with abnormal strain; and here we are compelled to assume some weakness in the valve tissues by which their nutrition has been modified. It may be that the mitral vela become chronically inflamed from the strain to which they are normally subject, from some cause which may be covered by the convenient cloak for ignorance, yclept idiosyncrasy; or that from some prolonged exertion overstrain, with consequent nutritive disturbance, has been set up; but the evidence thereof is only furnished at a late period when the connection with the exertion is not very palpable. The relations of chronic valvulitis with overstrain are such as to be most suggestive as to the line of treatment to be pursued and the value of rest. The occurrence of mitral disease, secondary to aortic disease, favors the hypothesis of strain being a cause of mitral valvulitis.

As to the anatomical characters of chronic valvulitis, Rosenstein ("Ziemmsen's Cyclopædia") describes them thus: "The anatomical and histological changes are induced by hyperplasia of the connective tissue, which in the chronic form produces a tough, firm, and fibrous tissue. The production of the tough connective tissue is confined sometimes more to the borders of the valves, at other times more to their insertions, but may often spread over the whole valve, and always has a strong tendency to shrinking. In proportion to the duration of the process the border retracts and shrinks more, and the valve becomes stiff and its motion impeded. The chordæ tendineæ are almost always involved in the process, and as their structure is exactly similar to that of the endocardium, they undergo the same thickening, fatty degeneration, or calcification. And from these tendons the process often spreads to the interstitial tissue of the papillary muscles." It is this shrinking of connective tissue, whether in the cicatrix of a burn or in a mitral valve flap after acute endocarditis, it matters not, that constitutes its evil property. The presence of

a certain amount more of connective tissue in a valve flap in itself would be no evil, indeed it would strengthen it, if it were not for this invincible contracting tendency. It is this contraction which leads to the distortion and mutilation of the mitral valve. It produces two distinct changes, known as stenosis and insufficiency. In the first the free edges of the valve become glued together, and the vela may be converted into a rigid curtain betwixt the auricle and the ventricle, a mere slit marking the ostium, known as "the buttonhole mitral;" or the vela may be soldered together, forming a cone projecting into the ventricle. In each case stenosis is the result. At other times the growth extends down the chordæ tendineæ, destroying them and fusing the free edges of the valves down to the stumps of the musculi papillares, so that the vela no longer arrest the backward flow of the blood on the ventricular systole. Sometimes one mitral flap is more affected than the other, and at other times both are implicated. This matted mass of tendon, muscular aponeurosis, and pathological connective tissue may be infiltrated with lime salts, and so become calcified, or, in other phraseology, ossified, as not uncommonly happens in the aged, or may be the seat of fatty degeneration. At other times the disease seems to pause, and even a species of repair is attempted in some cases. "The points or flaps are so large in proportion to the diameters of the ostia that a slight shrinking generally produces no insufficiency, and even a more extensive degree can be compensated by expansion of the unaffected part of the valve." (Rosenstein.) Sometimes the stretching of a portion of a valve flap is so marked as to constitute an aneurism. Of the mitral valve, more than any other valve, it is necessary to bear in mind the fact that all the cardiac valves can undergo some amount of disease and contraction before they become insufficient to close the ostium. The free edges of the mitral valve may be thickened and somewhat shrunken even without regurgitation or murmur, so long as the ostium is of unchanged size. Thus, temporary dilatation of the heart may reveal an insufficient mitral valve, of which there is no evidence, either objective or subjective, when the heart is of its normal size.

Chronic valvulitis of the aortic orifice is also a common malady. The chief cause of aortic valvulitis is admittedly strain. As we

saw in the Chapter on Hypertrophy and Dilatation, the aortic valves are often found diseased where there is a history of high arterial tension, and the muscular walls of the left ventricle are hypertrophied. The hypertrophied ventricle expels its contents into the arteries with abnormal force, the elastic vessels are over-distended and rebound violently, driving the aortic valves together with unwonted force, the consequence of which is that their nutrition is disturbed, and a growth of connective tissue follows which alters their form.

We saw in Chapter V how great and sustained muscular exertion, as in strikers, leads to aortic valvulitis; and also its production from the high-blood pressure in the arteries in chronic Bright's disease. Hilton Fagge has found spinal deformity, obstructing the blood flow in the aorta, a cause of aortic valvulitis, as Rokitansky found it a cause of hypertrophy of the muscular walls. The mass of evidence we possess as to the effects of strain in producing disease of the aortic valves is such as to render doubt no longer possible. Consequently, we find aortic valvulitis associated with and accompanied by changes in the muscular walls before such muscular changes are the consequence of the aortic disease, though of course, when aortic disease is established, further changes in the walls are induced. Muscle can grow stronger and larger by development of new fibrillæ, but the fibrous portion of the valves is strengthened by a growth of connective tissue, which, unfortunately, carries with it its shrinking or contracting tendency. When, then, the demand upon the heart is great and its nutrition is increased, muscular hyperplasia is accompanied by a growth of connective tissue corpuscles in the valves, which often is so pronounced as to constitute a deforming valvulitis. But it may be questioned if this nutritive change is necessarily always so excessive as to constitute disease. Dr. Wynne Foot, of Dublin, has recorded a case of enlargement of the right side of the heart in a case of cirrhosis of the lung, where, along with great muscular enlargement, the valves of the pulmonary artery were thickened and corpora Arantii developed, so that they resembled ordinary aortic valves, and yet there was no disease. The nutrition had remained normal; too often it runs riot, and then we get distinct valvulitis.

As to the changes which go on in aortic valvulitis, Hayden says: "The base of the valve-segments is usually the primary seat of these changes, as remarked by Corvisart, and next in order the corpora Arantii." From these points the morbid process gradually extends over the entire valve. I have attempted to make the subject a little more definite, and after careful study of many preparations in various museums, venture to hazard the following statement as being near the truth at least. When the valvulitis tends towards aortic stenosis, the growth commences at the base of the cusps, and from thence spreads towards the free edges; and, synchronously, there is a growth of connective tissue corpuscles in the arterial conus, so that there is stenosis of the conus along with obstruction due to the stiffened valves. This is the form usually seen in elderly persons with aortic disease. Common earthy salts are deposited in these valves till they become stone-like, rigid, and calcareous; so that the aortic orifice may be converted into a triangular opening with unyielding valves,—often rough from the growth of nodules, which in time become calcareous. The ostium has been found so small that it would only admit a bristle.

On the other hand, in young subjects the growth commences in the fibrous structure of the free edge of the cusps, extending from the corpora Arantii to the insertion of the cusps in the aortic wall, so that the free edge is thickened, and then in time contracted; consequently the cusps become insufficient, and there is regurgitation through them on the aortic recoil. It seems that along with these valve changes there is some dilatation of the arterial conus, and the aortic orifice is enlarged, so that there is an abnormally large orifice with shrivelled valves. But betwixt these pronounced types of disease there are many intermediate forms, where there is some obstruction and some regurgitation.

Aortic valvulitis is a disease of males rather than females, a fact which is so far corroborative of the hypothesis that strain is the great factor in its etiology. Aortic stenosis is comparatively rare among women, and is probably confined to those who are the subjects of chronic Bright's disease. Aortic regurgitation is less rare in women, though they are comparatively free from it. Bamberger found of fifty cases of aortic insufficiency, thirty-eight

were in men and only twelve in women. Yet in Vienna women do much of the hard and coarse work, and therefore are liable to aortic valvulitis. Of these fifty cases, no less than fifteen were under thirty years of age. On the other hand, three cases of aortic valvulitis have come under my notice in girls, one in a post-mortem on a girl of twenty-two, of whom I got no history of any value. The second occurred in a splendid girl of nineteen, with an excellent family and personal history, where there is an account of an acute illness referred to the heart, with the development of an aortic murmur. The other cases were also in fine girls. The only explanation at all apparent is, that acute aortic valvulitis is found of idiopathic origin, and that chiefly in girls. Dr. Peacock holds that congenital malformation of the aortic valves often "lays the foundations of the subsequent disease," having found such malformation in a large proportion of cases of aortic disease.

Statistics have been made as to the comparative frequency of disease at the various valves. My experience would place in order of greatest frequency: 1, mitral; 2, aortic; 3, tricuspid; and lastly, 4, pulmonary valvulitis.

There is no comparison betwixt the frequency of disease in the valves of the left and right sides of the heart. The greater muscular power of the left ventricle, and the greater pressure on the aortic and mitral valves, sufficiently account for this. In intrauterine or fœtal life the right side of the heart, which then bears the brunt of the circulation, is more subject to disease, incomparably more, than the left side. Disease of the pulmonary valves is all but unknown in adults; in hearts where all the other orifices are extensively diseased small festoons of tiny atheromatous nodules along the edges of the valve cusps are all that are found at the pulmonary valve. Primary tricuspid disease is scarcely ever found except in cases where the blood-flow in the pulmonic circulation is impeded. Tricuspid disease is almost always secondary to mitral disease, when the blood pressure in the pulmonic circulation is greatly raised, and the muscular walls of the right heart are greatly enlarged. This bears strongly on the subject of strain being the cause of cardiac valvulitis. Tricuspid disease is almost invariably regurgitant; there

is enlargement and dilatation of the ostium, with shrivelling and contraction of the valve flaps. Tricuspid stenosis is very rare, and when found is usually associated with mitral stenosis, and "in the great majority of cases the mitral lesion has been in excess, and in point of time manifestly in advance of the tricuspid." (Hayden.)

Disease of the pulmonary valves, this authority says, is of extreme rarity. Three cases were published by Dr. Ormerod, in two of which it is stated the other valves were healthy. The pulmonary valves may be pressed upon by diseased lung or cancerous growth till a pulmonary murmur may be induced. The pulmonary artery and its branches may be rendered considerably atheromatous without the pulmonary valves being implicated. Pulmonary disease is almost always congenital or of intrauterine origin. Syphilis is supposed to be associated with such disease in a number of cases.

Aortic Obstruction.—It is not by any means the invariable rule to find disease of the aortic orifice either purely obstructive or purely regurgitant, but for diagnostic purposes it is found convenient to describe the two typical forms. When the valves are rendered rigid, a distinct systolic murmur, usually harsh and saw-like, is heard at its greatest intensity at the second right costo-sternal articulation. But there is a stage preceding the well-marked obstruction when there is no murmur, and all the evidence we have of aortic valvulitis being present, is a muffling or modification of the aortic second sound. Such muffling, with an hypertrophied left ventricle and corded arteries, would furnish suspicion, at least, that the cusps were in the early stage of valvulitis. But aortic obstructive disease is a matter of slow growth, and my experience of individual cases is scarcely long enough to say much, positively, of the indications which precede the formation of a murmur.

When the murmur is developed, it is heard over a large area, sometimes quite loudly over the whole anterior surface of the thorax. It may be heard at the mitral area so distinctly that, if the stethoscope be applied there first, the impression made is that the murmur must be connected with that orifice. But if the stethoscope be carried to the aortic area, it will be heard louder

and still more distinct, until its point of maximum intensity is reached. From thence it will be found to be carried along the great arteries, often with much distinctness; it may in some cases be heard all along the dorsal region, and even on the right side. (F. Roberts.) Care must be taken to discriminate it from the murmur of a dilated and roughened aorta, which resembles it in area and in time. If the murmur be due to an atheromatous aorta, it is well to make the patient lean the head forward, when the finger in the sternal notch will often detect the "heave" of the diseased aorta. Such disease of the aorta is, however, commonly found along with aortic valvulitis. Then hæmic murmurs were once held to be aortic, but now they are usually referred to the pulmonary orifice. Beside the murmur, there are the evidences of hypertrophy of the ventricle, which is never, almost, dilated, but purely hypertrophied. The apex is lower than in health, and the ventricle is elongated. The apex impulse is strong and the blow determinate, but there is not the extensive heave of aortic regurgitation. A thrill may be present with a systolic aortic murmur, and the murmur is at times musical. (The musical note is explained as being due to a thread of fibrin hanging from an aortic cusp and vibrating in the blood stream.) The pulse is hard, wiry, and incompressible; "hardness and force signify hypertrophy behind the narrowed orifice." (Walshe.) But the pulse may vary according as great hypertrophy may coexist with a very narrow orifice, and so the ventricular force be broken, and its significance must be measured by comparison with the other evidence. As to rhythm, it is usually regular and even, but at times halts and intermissions are found. In two cases which have come under my notice, prolonged intermissions occurred.

The associations of aortic obstructive disease are such as to render its diagnosis usually unmistakable. In time and area it is identical with a hæmic aortic murmur. But aortic obstruction is a disease rather of men, at or over middle age, and is accompanied by hypertrophy of the heart, and thickened arteries, while the hæmic murmur is associated with a soft compressible pulse with general anæmia, and is usually found in girls. The pulse, too, is very suggestive; in obstruction it is usually slow and

steady, and not easily obliterated by pressure of the finger, while the hæmic murmur carries with it a pulse fast, feeble, and unsustained. According to Hayden, the hæmic murmur is not heard in the carotids in the recumbent posture. As the hæmic murmur is now recognized as being generally pulmonary, and conveyed to the left of the sternum, this fact, along with the general concomitants of the murmur, will usually suffice for a correct diagnosis. The murmur of aortic obstruction is often accompanied by a diastolic murmur from some regurgitation through the injured valves, but by no means necessarily so.*

A difficulty as to whether a murmur is mitral regurgitant, or aortic obstructive is not likely to occur with a practiced observer. But all practitioners are not adepts at the interpretation of physical signs, and it may be desirable to point out the distinctions. They are both systolic in time, but there the resemblance ceases. The area of maximum intensity of the aortic obstructive murmur is over the area of the right auricle, and conducted along the great arteries; there is pure hypertrophy of the left ventricle, and there is no accentuation of the pulmonary second sound; the pulse is steady, firm, and regular, and the patient is usually a man of middle age with thickened arteries. Then in mitral regurgitation the murmur is heard at the left apex away to the nipple, and even behind it; at the aortic area it is diminished in intensity, if heard at all; the left ventricle may be enlarged, but it is not pure hypertrophy; there is accentuation of the pulmonary second sound with enlargement of the right heart; the pulse is compressible, irregular in volume, and in the later stages in time too. Then, aortic obstruction has comparatively no subjective symptoms till the last stage of cardiac failure from defective nutrition of the walls is reached, while mitral insufficiency, if marked, is rarely without them, even in the early stage.

Older writers held that the risk of apoplexy from hypertrophy of the heart was much lessened when the current of blood was broken by aortic obstruction; this is obvious. Acute attacks of cerebral anæmia, closely resembling ordinary syncope, are found in some cases of aortic obstruction. In one case such attacks

* The points which distinguish it from the murmur of congenital disease will be given in Chapter XVI.

were readily induced by any physical exertion, especially if accompanied by mental anxiety. Treatment directed to the heart quickly relieved the head symptoms.

As to prognosis it is usually fair as to life, nor is there great liability to sudden death. If the hypertrophy and the stenosis be fairly proportioned to each other, so that a normal bulk of blood is passed through the narrowed orifice in a normal time, the individual is usually unconscious of anything being the matter with him, and the morbid change is only discovered by accident, as by examination for insurance. Nothing will be said further here as to the prognosis of aortic obstruction, as the subject will be comprehensively handled, and the prognosis of the different forms of heart disease compared in the final chapter.

Aortic regurgitation is distinguished by a murmur that is diastolic in time, pulmonary regurgitation being practically unknown. As this form of heart disease is very common, and of a most serious character, its diagnosis is a very important matter. It is accompanied by great enlargement of the left ventricle, and presents a distinct array of signs. On inspection, a large and forcible impulse extending over a wide area, considerably below the normal apex beat, is observed. Then a distinct and forcible thud can be felt. There is rarely any thrill. (Walshe and Hayden.) On percussion, the area of complete dulness is great, extending downwards and to the left. The heart is not so pointed as in aortic obstruction; it is more globular, and presents the characters of hypertrophy combined with dilatation. Auscultation tells us that there is a strong muscular wall (until the last stages of fatty degeneration are reached), but the indication *par excellence* is the loss of the aortic second sound, and the substitution of a murmur for it. This statement requires the following modifications for absolute accuracy. When the lesion is small, the sound produced by the closure of the aortic valves may still be heard, though there is a diastolic murmur at the base of the heart; at times the closure of the pulmonary valves may be heard through the aortic murmur, especially if it be low and soft in character. Also the aortic second sound may be accentuated, or blowlike, or muffled from thickening of the free edges of the semilunar cusps, before the stage of defined valve lesion is reached. But usually when

seen the case has reached the pronounced stage. A murmur is heard which is loudest at the base of the heart, at the point of the attachment of the third costal cartilages to the sternum. It runs down the sternum to the right apex, and terminates, often abruptly, at the ensiform cartilage. As to character it is variable; it may be low or loud, or it may be soft or harsh. Where the valve cusps are rough it is loud and harsh; there may be great regurgitation with dilatation of the aortic conus, and comparatively smooth-edged, shrivelled valves with a low, soft murmur. Consequently, mere loudness in itself is of little moment, nor is a soft murmur necessarily comforting. It is the most constant of all cardiac murmurs in consequence of its causation, and, Walshe says, "Aortic regurgitant murmur is, so far as I know, constant, where its physical cause exists; weakness of ventricular action obviously cannot have the same effect in rendering a regurgitant, as a direct murmur, obscure." Yet W. T. Gairdner has recorded a case where a regurgitant murmur ceased to be audible, and in double aortic murmur sometimes the obstructive portion is the more pronounced, while at other times the regurgitant is the prevailing character of the murmur. Still, generally speaking, aortic regurgitant murmurs are constant in character. Where there is also an obstructive systolic murmur, this is heard along the arteries, but the regurgitant murmur is not so conveyed, though authorities are not agreed upon this point. Walshe says there is no "aortic *blood murmur* diastolic in time;" once he found a diastolic murmur in displacement by pleuritic effusion. The area and time, then, of a regurgitant aortic murmur distinguish it from all other murmurs.

It seems possible even to diagnose which aortic cusp is torn down, when regurgitation is the result of the rupture of a cusp, if the requisite qualities for its recognition are also present. Balthazar Foster, of Birmingham, performed one of the most brilliant feats in diagnosis when he distinguished that the posterior segment was the one torn down in a case of traumatic rupture.* The regurgitant current was directed into the mitral current, and so the murmur of mitral stenosis simulated. He writes: "An

* Clinical Medicine, p. 127–8.

aortic diastolic murmur propagated to the heart apex usually means incompetency of the posterior (or mitral) aortic segment. I believe we may also say that a similar murmur, propagated towards the ensiform cartilage, indicates incompetency of either the left coronary or the right coronary segment, by which the regurgitant current is thrown more upon the septum of the ventricles." The practical value of this diagnostic refinement was shown by the prognosis given, to which reference will be made again in Chapter XVIII. The diagnosis was verified by the post-mortem. Few opportunities can occur to any one of making such observations, but they tell us clearly how accurately significant an aortic regurgitant murmur may be.

Then the pulse of aortic regurgitation is very characteristic. The blood thrown into the arteries by the ventricle at once enlarged and hypertrophied, causes rapid and great distension of the elastic vessels, while the aortic insufficiency permits the blood to escape backwards quickly, instead of passing slowly out by the arterioles. Consequently the pulse is "collapsing;" it has also been termed "jerking," and "water-hammer," or, again, "Corrigan's pulse" (from his memoir on "Permanent Patency"). It has been described as "balls of blood shot under the finger." This character is more pronounced when the hand is held up, and therefore generally noted in hospital in-patients rather than in other patients. (Broadbent.)* This pathognomonic pulse points to the nature of the lesion before the chest is examined. Aortic regurgitation is often followed by secondary mitral regurgitation, and then the pulse is modified accordingly; irregularity in volume being added, and the acuteness of the distension being less pronounced. The accuracy with which Dr. George Johnson diagnoses combined aortic regurgitation with mitral regurgitation, from the character of the pulse, is well known. The characteristics of this pulse are modified by the coexistence of aortic obstruction. It is not pronounced in the early part of the vulvulitis, and "indicates extreme inadequacy of the aortic valve, and not mere leakage, and as a guide to treatment it is less valuable than diastolic murmur, because developed at a later period."

* Sibson states that the collapse of aortic regurgitant pulse may be heard by placing the patient's wrist to the ear.

(Hayden.) When there is traumatic rupture of an aortic cusp the peculiar pulse is soon developed.

This rapid and excessive distension of the arterial system can be felt down to the anterior tibial, and seen in the retinal arteries. The arterial blow may be felt very distinctly, and Sir Thomas Watson relates a case where, for five years, the wife of a man with aortic regurgitation felt uncomfortable when taking his arm in consequence of the unpleasant blowlike sensation of his pulsating arteries. The large arteries are commonly dilated and atheromatous, and a large heaving aorta can often be felt at the sternal notch. Quite commonly the jerking temporal artery can be noted by the naked eye.

The prognosis of aortic regurgitation is such that something about it may be said at this point, without referring all to the chapter on Prognosis. In the first place, the prognosis is bad as to time as well as to life. As said before it is a disease of hard work, and the left ventricle is usually hypertrophied to some extent during that time of the valvulitis which precedes actual insufficiency. It occurs in young persons, in whom all valve changes progress more rapidly than in older persons. The hypertrophy is massive (for reasons given in Chapter V), but it is short-lived.

This has been pointed out by Mauriac and Jaccoud. Hypertrophy is durable when the nutrition of the heart-walls can be maintained, as in the hypertrophy of chronic Bright's disease. But in aortic insufficiency the backward current permits of the blood escaping past the orifices of the coronary vessels, and so the blood supply to the heart becomes insufficient. Ordinarily the backward current eddies in the sinuses of Valsalva, and the coronary vessels are well filled, and the nutrition of the heart-walls is maintained. Consequently, as Balthazar Foster shows, the prognosis of a case of aortic regurgitation is influenced pronouncedly by the fact which cusp is torn. If it be the posterior cusp, behind which there is no coronary artery, which is ruptured, then the case lasts longer than when either of the other cusps is torn down, behind which there is a coronary vessel. The cusps tend to throw the blood into the coronary arteries. Then the atheromatous aorta loses much of its elasticity, and so the rebound, which fills the coronary vessels, is diminished; and be-

tween these two causes the hypertrophy of aortic insufficiency is short-lived. Fatty degeneration of the fibrillæ of the massive heart is soon instituted, and heart failure is not far distant. Death is often sudden from the ventricle becoming overdistended in diastole and failing to contract. In this form of hypertrophy there is no inherent tendency in the new muscular fibrillæ to undergo fatty degeneration; the causes why these new fibres are short-lived are obvious enough.

The course is commonly rapid. For a time work can be continued, but sooner or later, often in no long time, it must be relinquished. The patient is anxious, nervous, and terribly conscious of the working of his heart. The carotids pulsate very visibly, and this beating is characteristic when seen on each side of an anxious, wistful, and not rarely somewhat frightened-looking face. A little later he is in bed, propped up, breathing with difficulty, the irregular pulse telling of fatty degeneration gradually enfeebling the heart. Often a process of skin degeneration is found in the later stages of this malady. It is unctuous and fatty, alike to the eye and the touch.

The atheromatous temporal arteries may be observed plainly jerking. If the mitral valve be secondarily involved, then pulmonary troubles come on and end the case. At other times the termination is sudden, may be in sleep, or the appetite fails, and unconsciousness, gradually deepening, steals on, until the heart stops in diastole.

The prognosis is much affected by the amount of obstruction combined with aortic insufficiency in each case; the more the obstruction and the less the regurgitation the better for the patient, and the longer he is likely to live.

In many cases of aortic regurgitation the mitral valve becomes secondarily affected. This is due partly to the extension of the inflammatory process from the aortic valves to the aortic flap of the mitral, that small sheetlike valve fixed into the base of the aortic cusps, partly by the fact that, as Traube pointed out, the mitral valves are closed by the regurgitant current as well as the ventricular systole, and their period of rest being so disturbed and broken, imperfect or perverted nutrition follows. Also, too, the left ventricle becomes enlarged, and then the mitral ostium is

correspondingly enlarged, and thus the valve flaps may become incompetent to close it on the ventricular systole. As has been insisted on before, the mitral flaps, in perfect health, are rather more than competent to close the orifice, and may undergo a certain amount of contraction before they become incompetent. So when this enlargement of the ventricle and dilatation of the ostium is marked, the mitral vela may become insufficient, and yet not be very extensively contracted. A most instructive case of this nature is to be found amidst Dr. W. H. Broadbent's patients at St. Mary's Hospital. A lad of very active habits has well-marked aortic regurgitation, and as his ventricle enlarged a regurgitant mitral murmur developed. The lad was, after this, laid up for a week or two with rheumatic fever, and during this time the heart returned towards its normal size, so much so that the mitral murmur disappeared; the valve flaps were once more competent to close the orifice. After his recovery, with the resumption of his habits, the left ventricle again became dilated to its old size, and the murmur reappeared. When secondary mitral disease is thus developed a systolic murmur, usually of soft character, is found at the left apex, extending towards the left nipple, which can be distinguished from the aortic murmur. The pulse, too, is modified; it is less distinctly collapsing, and is commonly irregular in time, and still more in volume. Prognostically, the development of mitral valvulitis, secondarily to aortic regurgitation, is of bad omen.

Mitral Obstruction.—Here the mitral vela are soldered together so as to form a fixed curtain across the ostium; or are agglutinated into a fingerlike cone projecting into the left ventricle. Consequently there is an obstruction to the blood-flow at this point, and a murmur, often accompanied by a thrill, is heard, which murmur is presystolic in time, *i. e.*, it occurs during the auricular contraction immediately preceding the ventricular systole. It was at first termed a diastolic murmur, and was confounded with the diastolic murmur of aortic regurgitation. W. T. Gairdner first discussed this murmur fully in this country, and pointed out its presystolic, or auriculo-systolic character; since then it has attracted much attention, and Dr. Hilton Fagge pointed out the modification of the first sound heard therewith, which

approaches the second in character, and so often misleads. The best summary of the sounds is given by my colleague, Dr. James Andrew, in "The St. Bartholomew's Reports," vol. xiii, 1877. He says: "A well-marked presystolic murmur, indicating obstruction at the mitral orifice, has the following characters:

"1. It occurs during the latter part of the diastole.

"2. Its point of maximum intensity is a little within and above the apex beat; it fades towards the left axilla, and is not heard behind.

"3. A diastolic thrill is present over the left ventricle.

"4. The murmur has a peculiar, harsh, droning sound, and becomes louder towards its close, which is abrupt.

"5. It is generally difficult to distinguish an interval betwixt it and the first sound.

"6. The first sound is shortened and intensified.

"7. Reduplication of the first sound is of frequent occurrence.

"8. The second sound is very feeble, or even entirely absent at the apex.

"9. The second sound is intensified at the left base, and very frequently reduplicated.

"10. The physical signs of dilatation of the left auricle and right side of the heart are present.

"11. The murmur varies much in loudness, length, and pitch, may disappear entirely, or even be replaced by a systolic apex murmur. These changes are of common occurrence as the case approaches its termination.

"12. The pulse is often small, rapid, and very irregular."

Some comments on these points will make the matter clearer to the reader. The thrill is often absent, but when present is of great diagnostic value, and marks the case as one of stenosis. The alteration of the first sound is probably related to the diminution in size of the left ventricle in mitral stenosis (contrasting with the enlargement commonly found in mitral regurgitation). The direction in which it is conveyed is probably the point which will be of most service to novices in the art of auscultation; its point of greatest intensity is to the right and above the apex beat, and it is lost as the stethoscope is moved towards the nipple

or the axilla, and it is not heard behind. In these respects it differs essentially from the murmur of mitral regurgitation. Dr. Broadbent is of opinion that the murmur is longer than that of regurgitation; but when the tricuspid leaks then the obstructive murmur becomes shortened, because the long murmur is no longer feasible. When the presystolic murmur is replaced by a systolic murmur, then it is probable that there is a "button-hole" mitral. Here there may be both murmurs; the consequence of which is that different opinions may be given by different authorities, leading to confusion, and not rarely something worse, and yet each observer may have been correct as to the murmur heard at the time he was consulted. Consequently it is unfortunate that so much stress is laid at present upon the diagnosis of the two forms of mitral murmur, because the recognition of the exact lesion does not bear much upon either prognosis or treatment; authorities differing as to whether the prognosis of stenosis or regurgitation is the worse; and agreeing that in both cases the correct plan of treatment is to build up the right heart by the administration of digitalis, iron, etc.; indeed, to carry out the principles to be laid down in the chapter devoted to treatment. The day of the prominence given to the presystolic murmur will ere long be over. Then as to the pulse of mitral stenosis. All agree that it is small, and Dr. Andrew is in accord with Dr. Balfour, of Edinburgh, in saying that it is "very irregular." On the other hand, Walshe, whose diagnostic accuracy no one will call in question, states that the pulse of mitral stenosis is regular, a view taken by Dr. Broadbent and many others, including the writer. Probably the irregularity of the pulse is due, to some extent at least, to the changes of lung pressure upon the enlarged and dilated auricle and right heart, and so may vary in different individuals. In the last stages of mitral disease the irregularity defies all rules. (Broadbent.) This discrepancy betwixt different writers as to the character of the pulse in mitral stenosis illustrates how different may be the experience of individuals; for Drs. Andrew and Balfour are well known for their care and acumen in diagnosis, and so, also, is Dr. Walshe, who writes ("Diseases of the Heart," 1873, p. 370), "And it must not be forgotten that mitral stenosis tends to equalize the irregular pulse of

mitral insufficiency." The lesson taught by this discrepancy may be usefully applied to some other refinements of diagnosis—that is as regards toleration of the opinions, and even apparent mistakes of others.

Mitral Regurgitation.—In mitral regurgitation the valve flaps are either contracted, and even fused down to their musculi papillares in advanced cases, or the regurgitation may be due to dilatation of the left ventricle and enlargement of the mitral ostium, so that the valve flaps become insufficient to close the ostium on the ventricular systole. In this last case some diminution of the size of the vela will be found present in nearly all cases. At times it may be due to irregular action of the musculi papillares in what is known as the "dynamic" mitral murmur. Consequently this regurgitant murmur is systolic in time. It is heard at the left apex, but is conveyed distinctly towards the left nipple, and may be heard of maximum intensity behind the left nipple. When well developed it is heard at the spinal column, at a point corresponding to the nipples. It is a less limited murmur than that of mitral stenosis, except when dynamic. It is usually soft in character, but may be loud. A very soft, almost inaudible murmur may go with great patency, and so be of the worst omen. On the other hand, a very loud murmur may be produced from a roughness of the auricular surfaces of the mitral flaps, and the regurgitation may be practically of little or no moment. When the tricuspid fails the blood current may be too feeble to elicit a murmur. At other times, where there is great dilatation of the left ventricle no mitral murmur may be discoverable (when there is every reason to suspect mitral leakage), until the treatment has strengthened the muscular walls, and then a mitral systolic murmur is distinctly heard. The amount of regurgitation, as Skoda pointed out, is rather to be measured by the accentuation of the pulmonary second sound than by the loudness of the murmur itself. In all mitral disease there is obstruction to the flow of blood in the pulmonic circulation, with enlargement of the right heart, and so the pulmonary valves being closed with abnormal force, produce a louder sound, and the diagnostic value of accentuation of the sound produced by closure of the semilunar valves is very great. It does not follow that because there is no increase, but

even a diminution in the blood pressure in the systemic circulation, that therefore the blood pressure in the pulmonic circulation should not be increased, and that, too, without a mitral lesion—where a great deal of increased stress is thrown on the right heart through the large capillaries of the pulmonic circulation. To this accentuation of the pulmonary valve sound must be added the evidences of right-side enlargement, and often fulness with pulsation of the veins of the neck. When the right-side enlargement is insufficient or failing, then there are evidences of lung engorgement, or stasis, with embarrassment in breathing, and the characteristic "heart-cough." There is asthenic congestion of the lungs, and general enfeeblement with dyspnœa readily induced. This *rückwirkung* is common to both forms of mitral disease. At times there are attacks of hæmoptysis, and in elderly persons, especially in mitral regurgitation, there is often persisting bronchorrhœa. Indeed, mitral stenosis is most common in young subjects, while mitral regurgitation is found alike in young and old. If there is found a mitral murmur in an elderly person, it is almost always a regurgitant murmur. Then, too, in mitral regurgitation there are evidences of change in the left ventricle, as pointed out in the last chapter; it is enlarged commonly by combined hypertrophy and dilatation. "In mitral stenosis this chamber is very generally found to be small, and its muscular substance is no thicker, and may, perhaps, even be thinner, than under normal conditions. The aorta, too, is often small and thin-walled." (Hilton Fagge.) On the view of hypertrophy put forward in Chapter V, this is quite intelligible—there is no increased distension of the left ventricle in mitral stenosis. The same writer says further: "But in some cases of mitral stenosis, and in almost all cases of 'mitral regurgitation,' the left ventricle is large and fleshy, and not infrequently it is as much dilated and hypertrophied as in aortic regurgitation." This is quite in accordance with the view of increased internal pressure as the cause of the enlargement of the left ventricle in mitral regurgitation. In the cases of left ventricular enlargement there are the muscular changes subsequent to the valvular lesion. But in some cases of mitral regurgitation, as well as of mitral stenosis, there are the changes in the ventricle previous to, or co-

existent with, the valve lesion to be considered. As said before, mitral lesions are common in the subjects of chronic Bright's disease, and the muscular hypertrophy, and the high blood pressure in the arteries, set up mitral valvulitis from overstrain. Speaking broadly, then, in mitral stenosis there is no secondary enlargement of the left ventricle, while in mitral regurgitation this is common.

When a mitral murmur, systolic in time and regurgitant in character, is found along with other evidences of disturbance of the vascular system, it may fairly be diagnosed as of organic origin, and indicating insufficiency of the mitral valve flaps of some kind. But there are mitral systolic murmurs which are not organic, but "hæmic" or "dynamic." Dr. George Balfour calls this "curable mitral regurgitation;" and states that it is found in conditions of muscular relaxation and debility (involving the ever-acting heart), and of blood alteration. He agrees with Naunyn that this is not an arterial murmur. He says: "But it is a still more curious fact that, by carefully examining all the cardiac area, we discover that the position of maximum intensity of this so-called arterial bruit is not over an artery at all, nor in any of the usually accepted areas of cardiac sounds and murmurs, but is actually about one inch and a half, or rather more, to the left of the pulmonary area, and in the same plane immediately over the part where the appendix of the left auricle pops up from behind, just to the left of the pulmonary artery. This so-called arterial murmur is, therefore, not arterial at all, but strictly auricular in its source." Improvement of the blood and of the nutrition of the heart-walls causes the murmur to disappear. Other mitral murmurs, which are systolic in character, also disappear in time, as in chorea, for instance. Several such dynamic murmurs have come under my notice, which have followed sustained effort, like a long enforced run, in boys; they have had no accompanying evidences of organic change, and disappeared after a time. One loud presystolic murmur is known to me which is not due to disease. It was pronounced not to be serious by the late Dr. Latham, twenty-five years ago; and during a very energetic life there have been no evidences of its having an organic origin; yet it is very loud.

In mitral regurgitation the pulse is often quite characteristic. It is irregular alike in time and in volume. A full beat may occur with small ones, or a small beat among normal beats. Full beats and small ones may be found together, especially where there is also great dilatation of the walls of the left ventricle. We may suppose that the amount of blood escaping backwards through the mitral leak at each ventricular systole is not always the same, and also that the contractions of the ventricle are not always absolutely alike, nor an identical amount of blood thrown into the aorta at each ventricular systole. Irregularity in volume is more indicative of mitral regurgitation than is irregularity in time; the latter is rather significant of dilatation. Dr. Broadbent thinks that the irregularity of mitral regurgitation is due often to changes in pressure during respiration, more markedly at the end and commencement of the respiratory act, especially in the early stages. In the later stages the irregularity defies all rules. He thinks, in mitral stenosis, irregularity of the pulse is found only in the last stages, and is then more marked in the pulse than in the heart. The impulse in mitral regurgitation is, he thinks, a "tap" rather than a "heave."

The irregularity of the pulse in mitral regurgitation is often so characteristic as to enable a diagnosis to be formed before the chest is auscultated, and frequently gives a direction to the investigation of the physical signs that is very useful.

When it is blended with aortic regurgitation the resultant compound is sometimes perplexing; but a little experience and reflection will soon enable the reader to master the difficulties and separate the two factors, (1) the collapse, (2) the irregularity, without much hesitancy, at least in most cases. The pulse is at once feeble, small, often irregular, and collapsing, characteristics which are rendered more pronounced by holding up the arm.

The backward processes set up by mitral disease, especially after the tricuspid is secondarily affected, are given in Chapter IV, which must now be referred to and read again. The prognosis of mitral disease is a matter yet in dispute as to the chances of life. Some assert that stenosis is more favorable to longevity; others hold that regurgitation is more compatible with a prolonged existence. The matter will be discussed in Chapter XVIII.

One thing may be asserted of mitral disease, there is no marked tendency to sudden death with it, but rather to slow exhaustion from some of the resultant sequelæ—the heart, though the cause of death, is not itself always the first to die.

The treatment of mitral disease must be conducted on the principles to be laid down in the chapter devoted thereto. All authorities are agreed that digitalis is useful in mitral disease. The difference of stenosis or regurgitation is little, as regards the treatment of mitral valvulitis. The resultant consequences are the same, except that Dr. Broadbent thinks that ascites is more common in mitral stenosis than in regurgitation.

Disease of the Pulmonary Valves is very rare, except as a congenital defect. Rosenstein has seen a case, and says, that Speer had a case where the valvulitis had extended so far that it was impossible to pass a quill through the orifice. But a pulmonary murmur is a very common occurrence. It is heard at the third left costo-sternal articulation, and is heard up to the clavicle, not quite in a direct line, but slightly outwards towards the shoulder. It is not heard so well on the right side, and is not conducted along the aorta or arteries. Such is the murmur of pulmonary stenosis as observed by Elliotson and others. It is not heard at the apex or the back. In the rare event of such disease being met with, there would be the evidences of right-side enlargement (p. 127). In a case under care at present there is no cyanosis, but the patient is very small for his age. Pulmonary murmurs may be produced by pressure on the vessel by masses in the pericardium, or tubercular disease of the left lung. Eight such cases have been recorded by Dr. Da Costa; in seven of these the murmur was very localized, and heard over a very small area. At other times the pulmonary artery is not covered by lung, and then a murmur is heard; in one case under care a very loud, harsh murmur can be heard over a considerable area. This murmur is not simply the hæmic murmur of anæmia, but is pronouncedly affected by full inspiration, when it is heard much louder; and by full expiration, when it is much diminished. The hæmic murmur is usually soft, and is accompanied by the *bruit de diable*, and by the evidences of general anæmia.

A localized murmur heard at the pulmonary area, and accom-

panied by evidences of right-side enlargement, may, however, not be due to pulmonary disease. Walshe, in his last edition, quotes such a case reported by Dr. Eddison, of Leeds, and myself. On post-mortem examination, tiny festoons of atheroma were found on the pulmonary cusps, which were perfect, and in no way injured by the commencing atheromatous process. But the aortic orifice was very much diseased, and there was a "button-hole" mitral. The blood current was so broken at the mitral orifice that a very feeble aortic murmur was produced, heard over a limited area; the right-side enlargement was the usual result of the mitral disease.

The error was a very natural one, and may easily be made by others.

A systolic pulmonary murmur may be heard just before death, in cases of the ante-mortem formation of a clot in the right ventricle.

As to pulmonary regurgitation it is practically unknown. From experiments on animals, it has been ascertained that pulmonary regurgitation produces a murmur heard down the ventricle. In a case recorded by the late Dr. Warburton Begbie of pulmonary valvulitis, a loud, blowing, systolic murmur was audible over the upper portion of the chest generally; it was not transmitted into the carotids, and was loudest at the left third costal cartilage, near the sternum, where a diastolic murmur of much less intensity, and a thrill likewise existed. The pulse was seventy-two, regular, and not visible. The valves of the pulmonary artery were four in number, and incompetent. The other valves were healthy. The man died from an accident. (Hayden, p. 1000.)

The pulmonary artery may be thickened and atheromatous from overstrain in mitral disease or pulmonic obstruction; but even then the pulmonary valves are rarely more than slightly affected.

Tricuspid regurgitation is a common consequence of mitral disease. When the right side of the heart becomes enlarged and thickened, more strain is put upon the tricuspid valves, and then valvulitis follows. Older writers used to speak of a sympathy existing betwixt the mitral and tricuspid valves as the cause of the secondary tricuspid disease. It is found when the right side

of the heart is enlarged, let the cause be what it may. Tricuspid regurgitation is not, however, always the result of valvulitis, but may occur from dilatation of the tricuspid ostium. The late Wilkinson King, in a most interesting essay, tried to make out "a safety-valve action of the tricuspid," and held that leakage of the tricuspid valve was useful in cases of engorgement of the right heart. In the hearts of diving mammals, and in animals whose safety lies in flight, there are special arrangements (the moderator band of Reil) to prevent tricuspid regurgitation. Venous pulsation is often found in right-side engorgement; but this regurgitation probably depends on insufficiency of the valves. King found the tricuspid valve to leak readily when the right side of the heart was distended. Tricuspid regurgitation without valvulitis must be admitted; but in the bulk of cases there is some valvulitis, just so much that the valves are still competent when the right ventricle is not very distended. Rokitansky found stretching of the valves usually to accompany dilatation of the muscular chambers, and Peyton Blakiston has found that in 126 cases where the tricuspid ostium was enlarged, in no less than fifteen "the valves had increased in size so that they perfectly closed the foramen during systole, and there was no regurgitation;" and Niemeyer writes: "When the ostium dilates, the valve grows in breadth and length, almost always remaining competent to close the widened orifice."

In tricuspid valvulitis the same pathological changes are found as in mitral valvulitis, except that tricuspid stenosis is very rare, and the change set up is almost invariably that of insufficiency. The valve flaps are contracted, and the chordæ tendineæ shortened. The small valve of the fixed wall may be shrivelled up till the tip of the forefinger can scarcely be inserted betwixt the valve and its wall.

The signs of tricuspid regurgitation are venous pulsation, evidences of right-side enlargement, and a systolic murmur heard at the ensiform cartilage. The murmur is soft in character, and heard over a small area only. It rarely masks the systolic sound completely; is of rarer occurrence than tricuspid regurgitation itself; is, probably, not always detected when it exists, and often escapes detection because it is covered by a powerful mitral mur-

mur, and in some cases is impaired in distinctness by a deep-seated venous hum. (Walshe.) The venous pulsation of tricuspid regurgitation is to be distinguished from that caused by the pulsation of the carotid arteries, according to Hayden, by the fact, "that the true jugular pulsation is partially arrested by collapse of the veins during *inspiration*, whilst it is exaggerated at the acme of expiration. The carotid pulse is in no way influenced by respiration." Jugular pulsation may be the result of the contraction of the enlarged right auricle, without any deformity of the valve flaps. There are, too, the general evidences of tricuspid failure (Chapter IV), as liver pulsation, congestion of the valveless portal circulation, and the rest.

Tricuspid obstruction is a very rare form of disease. When it occurs the tricuspid valves are united, and the edges of the orifice may be rough, and even calcareous. It usually occurs with disease of the other valves, aortic or mitral, or both.* A tricuspid obstructive murmur has been diagnosed by several physicians, as Rutherford Haldane, Balthazar Foster, and others. Foster found "the area of cardiac dulness increased laterally, a thrill most distinct at the ensiform cartilage, and a murmur which had its point of maximum intensity at the base of the ensiform cartilage, close to the right edge of the sternum. It began softly, but grew louder and harsher up to its end, which came abruptly with the first sound. The murmur was propagated faintly to the base of the heart, but was not audible in the axilla, or at the back of the chest. The tricuspid valve segments were adherent and formed a round orifice of communication between the auricle and ventricle, which admitted the forefinger beyond the first joint. The edges of the orifice were thick and rough on the auricular surface, and studded with calcareous spots. The aortic, pulmonic, and mitral valves were healthy. The orifices of the aorta and the left auriculo-ventricular aperture were narrowed, the latter only admitting the tips of the two first fingers."

The prognosis of disease of the right side of the heart is neces-

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* Niemeyer writes: "I have repeatedly observed contraction of the tricuspid with rupture of the chordæ tendineæ, as an accompaniment of severe stricture of the mitral." The combination of mitral and tricuspid disease has been described by Bamberger.

sarily bad. As long as the tricuspid valve holds the system is largely protected from the consequences of heart failure; but when it leaks the general consequences of blood stagnation are felt.

The premurmuric stage of valvulitis is a matter of growing interest. Probably we can know little of this in any but the aortic valves. Strain is the great cause of aortic valvulitis, consequently it is common in forms of toil where severe and general efforts are made. The first changes are those of hypertrophy of the left ventricle, with accentuation of the aortic second sound. Later on a regurgitant murmur is audible. In advanced stages in the subjects of lithiasis there are contracted arterioles, a tense pulse from high blood pressure in the arteries, and hypertrophy of the left ventricle. This hypertrophy maintains the high tension in the arteries, which in time become atheromatous. Such are the associations of disease of the aortic valves in the elderly. There are distinct changes which precede the murmur. Walshe, Hope, Fuller, and Da Costa all speak of a dull or clanging tone in the aortic valves when thickened, before a murmur is heard. Such change of tone occurring where distinct accentuation has been heard, will, probably, indicate that disease is progressing in the aortic cusps. The question, then, may be raised, "Are we always compelled to wait for the comparatively remote and brutal answer of a murmur before we can feel sure that the aortic valves are becoming diseased?" I firmly believe that a murmur is not essential to the diagnosis in every case.* This view did not escape the attention of an early observer, Dr. Clendinning, who held the view "that the valvular disease is rather the consequence than the cause of hypertrophy, as this latter condition of the heart, itself the result of a vital and not of any mechanical condition, is known to predispose to inflammation of all parts of the body, and to that of its own inner lining membrane, of course, amongst the number." (Joy, "Library of Medicine.") The phraseology is somewhat antiquated, but the fact remains that Clendinning thought hypertrophy of the heart was a cause of valvulitis.

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* The reader who is interested in this matter will find in the Edinburgh Medical Journal for February, 1879, a paper where this subject is discussed at some length, and the views of Dr. Broadbent, Dr. Sansom, and Dr. Stephen Mackenzie given, as well as those of the writer.

CHAPTER VIII.

DISEASES OF THE MUSCULAR WALLS—MUSCULAR CHANGES—MYOCARDITIS—FATTY ACCUMULATION—FATTY INFILTRATION—FATTY DEGENERATION—FALSE HYPERTROPHY—ATROPHY—BROWN ATROPHY—AMYLOID DISEASE—SYPHILITIC GUMMATA—TUBERCLE—CANCER—PARASITES—POLYPI.

THE question at times arises, is hypertrophy of the heart ever a disease *per se?* Older writers looked upon hypertrophy as the disease of the heart *par excellence.* On the other hand, my friend Dr. Angus Fraser, of Aberdeen, teaches that, broadly speaking, there is but one disease of the heart, and that is "Dilatation." There is much truth in this broad statement; the acute danger in heart disease is arrest of the heart's action in diastole; the chronic danger is dilatation. Still, under some circumstances, as we saw in Chapter V, hypertrophy may be a disease *per se;* but this is very rare. It is almost invariably a secondary consequence of something which precedes it in time.

The heart is of variable size in different persons. There are persons with naturally small hearts and feeble circulation. These are, according to the late Professor Laycock, persons of the strumous diathesis. On the other hand, in persons of the sanguine arthritic diathesis the bloodvessels are numerous, the heart is large and powerful, and the blood-corpuscles are numerous. The latter class furnishes the greater number of cases of organic disease of the heart; the former rather gives us the cases of functional derangement combined with adynamy.

It is of importance to recognize this fact of the variable size of the heart in different persons, as otherwise mistakes may occur, causing much needless apprehension and misery. For instance, a medical friend of mine has a large heart, a well-developed vascular system, and is a great athlete, especially remarkable for his endurance. His large heart beats slowly, just like an hypertrophied heart, because, as described in Chapter I, the roots of the

vagus are well flooded with blood, and the powerful heart is held back so that the blood in the arteries may have time to escape out of the arterioles before the next ventricular systole comes; otherwise the arteries would be overfull, and the consequences, early and remote, of overdistension of the arteries would follow. A well-known provincial physician of the old school pronounced this state hypertrophy, and for some years my friend was made uneasy; but as he grew no worse and his capacity to endure exertion remained undiminished, he began to distrust the opinion. On consulting me, my opinion was given to the effect that he had an unusually good and powerful heart; and that it was due to this that he was so remarkable for sustained endurance in the cricket-field and elsewhere. This was precisely the condition of the famous racehorse "Eclipse," who had a very large heart. His "staying power" was even more remarkable than his speed, and when he died at Lannes, in February, 1787, he was opened in order to see if any peculiarity could be found to explain his remarkable prowess. "The heart of the indomitable creature was found to weigh fourteen pounds, and Vial de St. Bel, who opened him, attributed his extraordinary and unflinching courage to the size and vigor of his huge blood-pump." When the famous Irish greyhound, "Master Magrath," died, his body was sent to Professor S. Haughton for examination. The animal had also been more remarkable for his endurance even than his speed. The heart was of unusual size and power, and Professor Haughton wrote, "I believe that Master Magrath's great heart (like the Nazarene locks of Samson's hair) was the real cause of his extraordinary running powers." Dr. Gowers thinks that this hypertrophy was the result of the long-continued exertion; but I venture to differ from him, and think the large heart the chief factor in the animal's great power. Certainly Dr. Gowers is right in stating that hypertrophy of the heart is sometimes the result of overexertion; and, as said in a previous chapter, such hypertrophy is usually found antecedent to aortic valvulitis. The articles on hypertrophy of the heart, on dilatation, etc., by Dr. Gowers in Russell Reynolds's "System of Medicine," are excellent. He but narrowly misses the explanation of the cause of hypertrophy. He says, "Increased blood pressure within the

heart during its systole is the common cause of its muscular overgrowth." As seen in Chapter V, it is the overdistension during diastole which excite strophic changes. Dr. Gowers, however, grasps the facts more clearly in the article on dilatation, where he writes, "The dilatation of the heart is produced in every case by its overdistension with blood. Just as the various causes of hypertrophy involve, as the efficient cause, overwork, so the various causes of dilatation involve overdistension. The immediate cause of this overdistension is, in each case, the existence at the end of the diastole of an endocardial pressure disproportioned to the resisting power of the wall of the heart, and before which the heart yields. The act of dilatation thus occurs during the diastole of the heart." As seen in Chapter V, the primary causes of dilatation and hypertrophy are the same. I should in that chapter have expressed my indebtedness to Dr. Gowers, if my views had not appeared in the first edition of this work (pp. 69, 70, 71), only not put so forcibly forward as in this edition.*

Hypertrophy is very rarely found as a disease *per se;* and a naturally large and powerful heart is not necessarily an abnormal or diseased heart. The relations of hypertrophy to the various forms of valvular disease are important clinically. We saw in the first chapter how, as the heart is developed, as evolution proceeds, its muscular power is economized by the formation of valves by folds of the endocardium, strengthened by fibrous bands. When the complex valves of the human heart are injured and rendered insufficient, the heart is degraded to a lower type—and its efficiency depends upon compensating muscular growth. If the muscular growth be sufficient to cause the normal amount of blood to be passed into the aorta in a normal time, thus perfect compensation is attained, which may last for many years. This is best seen in aortic stenosis, where a thickened ventricle compensates a narrowed orifice. Less perfect and durable is the compensatory muscular growth in the left ventricle in aortic regurgitation; but in those cases of mitral regurgitation

* In a recent thesis Dr. Rosenbach, of Breslau, ascribes the first step towards hypertrophy as a primary dilatation of the particular auricle or ventricle in diastole; thus quite agreeing with the view put forward by me. Centralblatt Med. Wiss., No. 37, 1878.

occasioned by acute endocarditis, and where the injury done is limited and exhibits no tendency to proceed, but becomes stationary, the compensation by muscular growth in the right heart often produces a new equilibrium which lasts for many years, and is not far below the normal equilibrium, if the demand upon the heart be not severe. If the demand be "levelled down" to the new equilibrium by a quiet life, the case may proceed without growing worse, and almost a normal length of days be attained. Several such cases—one reached ninety-two, another eighty-six years—were given by Dr. Andrew Clarke, in a paper read before the Medical Society on the prognosis of heart disease, December 17, 1877. In his work on dropsy, Basham says: "Disorganization of the valves of the left side of the heart is a well-known concomitant of acute rheumatism, yet it is not a little remarkable, and must be familiar to most physicians, how few suffer from dropsy of those whose mitral valves only have become disordered, and in whom a mitral murmur is easily detected. How many patients we meet with who for years have thus suffered from an imperfect mitral valve, who enjoy a fair share of health, and who, except that they are not equal to any rapid bodily effort, such as running, or being hurried in their movements, go through a life of average duration without other inconvenience." Dr. King Chambers, in his "Clinical Lectures," gives cases of "defective valves" of fifty-seven years' standing, of fifty, of forty-four, and twenty-seven years' standing. All mitral, "in whom no inconvenience to be called illness has followed."

On the other hand, where the vulvulitis is of the originally more chronic, sclerotic, or contracting variety, it constantly progresses, and the muscular compensatory growth has to keep pace with it until it can no longer do so, and failure by dilatation must, and sooner or later does, ensue. The muscular growth cannot go on indefinitely, and a time comes when the muscular compensation fails to meet the narrowing orifice, or the force of the regurgitating current. But the aspect of every case of valvular disease rests largely, as regards the prognosis, on the muscular modifications which accompany it, or follow it, and, consequently, upon the nutritive powers of the individual. If the mutilation of a valve be small and the mischief stationary, then a slight muscu-

lar hyperplasia is sufficient for a perfect and durable compensation, while in progressive or contracting valvulitis, the changes in the valves require constant muscular changes to keep pace with them, or the compensation fails.

The muscular changes which accompany or follow the different forms of valvulitis are as follows. Aortic stenosis leads to considerable hypertrophy of the left ventricle, without enlargement of cavity. Aortic regurgitation induces great enlargement of ventricular cavity (left), with massive hypertrophy. In mitral regurgitation there are "forward" as well as "backward" changes. The backward changes, enlargement and thickening of the left auricle, of the pulmonic vessels and of the right heart, are common to both forms of mitral disease. The forward changes are different. In regurgitation "the left ventricle is large and fleshy," while in stenosis "this chamber is very generally found to be small, and its muscular substance no thicker, and may perhaps even be thinner than under normal circumstances. The aorta is often small and thin-walled." (Hilton Fagge.) At times, however, in cases of mitral stenosis, the left ventricle is large and thick, but in such cases I incline to hold that the hypertrophy of the left ventricle preceded, and was the cause of the mitral valvulitis. In a previous chapter it is stated that a high blood pressure in the arteries, with hypertrophy of the left ventricle, puts more strain than normal on the mitral valve flaps on each ventricular systole, and so valvulitis is excited. A growth of connective tissue from the fibrous bands of the valve vela is a correlation of growth with the muscular hyperplasia. Thus we are compelled to admit that the muscular changes found with valvulitis may precede or accompany the valve changes, and not merely be the result of them.

Then again, valvular incompetence may be the result of dilatation of the cardiac chambers without valvulitis. Such a condition of the right heart is quite common at periods of acute engorgement of that chamber. In cases of rapid dilatation of the left ventricle, mitral insufficiency is so produced. One such case in a woman with severe menorrhagia was very marked, and reduction of the size of the chamber, and, with it, a diminution of

the mitral ostium, was followed by a disappearance of the evidences of regurgitation.

The remarks as to the relations of valvulitis to muscular hyperplasia in the left side of the heart, apply equally to right-side changes, and the association of tricuspid valvulitis with enlargement of the walls of the right heart is well known. Though the form of such valvulitis is commonly that of insufficiency, still tricuspid stenosis is not unknown.

Acute Myocarditis.

Rindfleisch commences his acccount of myocarditis by assuring us that it is the darkest chapter in all pathology. Inflammation of all muscular structure is but imperfectly understood, and such inflammation is rare by comparison with inflammation of other structures. Acute myocarditis is usually more or less general throughout the heart, and is found along with pyrexia, usually septic in character. When found along with endocarditis or pericarditis, it does not commonly extend far into the cardiac wall. It commences, according to Virchow, either in the muscular bundles themselves (*parenchymatous myocarditis*), or in the intermuscular areolar tissue (*interstitial myocarditis*). The inflammatory process may run on to the formation of pus, or purulent myocarditis. The structure of the heart is, at first, of a dark-red color, but later on becomes of a grayish-yellow, or grayish-red, with accumulations of pus in minute masses. Under the microscope the primitive bundles of muscular fibre become increased in bulk and swollen with fluid, and the cross-markings become indistinct. To the touch the heart-walls feel soft and boggy, and are easily torn, the structure being readily broken up into irregular fragments. When pus is formed the purulent accumulations often run together and form an irregular abscess, which may perforate the endocardium and open up into the circulation.

Such myocarditis is found commonly with pyæmia, where it is called metastatic, or with puerperal fever, typhus, low forms of scarlatina, or, according to Oppolzer, not rarely with cholera. Acute softening of the heart is a common cause of death in re-

lapsing fever; convalescents therefrom not uncommonly dropping dead when walking around the ward, from syncope due to the weakened condition of the heart. In what myocarditis differs from the changes which go on in muscles during a sustained high temperature, ascertained by the process of "harpooning," it is impossible to say. Acute myocarditis is rarely absent in fatal endometritis; but how far it may proceed, and yet be compatible with recovery, is not yet known.

The symptoms of acute myocarditis are not well marked. There is an absence of objective phenomena, as palpitation, while the subjective symptoms are those common to all cardiac failure. When found along with septic pyrexia, the failure of the heart's power is the most significant indication of the disease; while, if it be accompanied by ulcerative endocarditis, a murmur may be heard. Pain is not always present in myocarditis; its presence is of some value, but its absence proves nothing. Probably, extreme rapidity of the pulse, with great weakness in it, is the most reliable sign of acute myocarditis when extensive and diffuse. After death the blood is dark and of a violet hue; but there are no indications of such blood change in life.

As to the progress and termination of acute myocarditis, being rarely found alone, these depend largely upon its concomitants. It is very improbable that it is altogether absent in cases of sustained high temperature which recover; just as degenerative changes in other muscles are found by the process of harpooning to exist in patients who recover. The scars of Dittrich furnish testimony as to by-past disease, and are not rare in the dead-house. As to whether, in any case, acute myocarditis is present extensively or not; or whether its progress has not reached a point incompatible with recovery; or that point is irreparably passed; probably exceeds the diagnostic powers of most physicians. As to treatment little may be said. The condition of the heart must be looked at alongside the general state, and stimulants, beef tea, milk, digitalis, with or without strychnia, are indicated.

Chronic Myocarditis.

Chronic myocarditis is stated by German writers to be the great cause of degeneration of the muscular structures of the heart;

and Schrœtter says "that we must learn to consider a great number of the cases of fatty degeneration as only the result of a chronic parenchymatous inflammation." Such a process will certainly lead to a softening of the heart structures, but whether, what we understood by fatty degeneration of the heart, is the condition thus induced, may be questioned. The state reached by chronic myocarditis is rather that found in a case described by Hope.

An intermittent pulse had existed six years, and as the cardiac symptoms increased, the patient was advised to take exercise, with the idea of reducing the fat on the heart. This he did to excess, and the symptoms became aggravated. He travelled up to London, and then presented the following appearance and symptoms: "His complexion was very sallow, with purplish cheeks, nose, and lips; oppressive dyspnœa, but not orthopnœa; pulse extremely intermittent, irregular, and unequal, an occasional beat being stronger and larger than natural, while the intermediate beats were very small, weak, and often imperceptible. Slight œdema pedum. Auscultation.—Impulse, a flutter, with an occasional bound of inordinate force. Sounds both weaker than natural, and the first as short and flapping as the second. No murmurs. Contractions of the heart were 130 to 140 per minute, and the pulse 40 to 60. Percussion prevented by a vesication on the præcordial region. The symptoms increased. Whenever drowsiness slackened voluntary respiration, gasping came on and aroused him, and this occurred alternately every four or five minutes. Next supervened expectoration of dark blood, failure of the pulse, and moderate anasarca; finally diminution of sensibility, and death in a fortnight after a protracted struggle. Diagnosis.—Hypertrophy with dilatation, which I inferred from the occasional strong impulse, and strong large pulse. No valvular contraction or regurgitation, because no murmur, and because great mitral contraction or regurgitation was incompatible with the occasional strong beats of the pulse, which is always weak in such mitral disease. Softening, because without mitral disease, the pulse was irregular, etc., and because there was passive hæmoptysis and great venous retardation; also because the sounds were weak and the first short, though there was hypertro-

phy with dilatation. Pulmonary apoplexy. No hydrothorax. Autopsy.—Cavities of the pleura contained two ounces of blood. Lungs universally engorged and black; while inferior lobe of the left in the state of pulmonary apoplexy. Heart dilated to the size of a bullock's, being nearly three times the size of the closed fist of the subject. Walls of left ventricle thickened to about three-quarters of an inch; muscular substance dark red from sanguineous engorgement, and so much softened that a finger and thumb passed through it with very little pressure. All the valves perfectly healthy and capable of discharging their functions, except that the mitral and the aortic were strong and rather opaque, from hypertrophy of the fibrous tissue. Walls of the right ventricle of natural thickness, but the external third of the muscular substance was replaced, over a considerable extent, by fat. Auricles dilated to double."

Probably this case will give the symptoms and pathological condition of softening of the heart from inflammation, better than any mere array of general indications. Hope calls it a case of extreme softening.

Rokitansky thought the left ventricle the commonest seat of such inflammatory changes, and especially the apex; then the right ventricle, more rarely the right auricle, while the left auricle possessed almost complete immunity. Such softening may lead to an aneurism of the heart at the part affected. At other times an abscess may form in the softened tissue. In other cases, again, the injured tissue forms a sort of cicatrix, the scars of Dittrich, which are not uncommonly found in the dead-house. When this process is set up around the right or left conus arteriosus, and then is followed by a growth of connective tissue possessing contractile qualities, a band of cicatricial tissue is formed; which constitutes an obstruction to the blood-flow, and is known by the name of the true cardiac stenosis of Dittrich. Where the inflammatory process produces merely a softening of the tissue of the part affected, then a bulging, or true cardiac aneurism, is formed. This is most commonly found at or near the left apex, but it has been found in the septum ventriculorum. It may vary in size from a bean up to the size of the heart itself. The visceral pericardium stretched over it may become almost lost and

invisible; while the endocardium lining it may become atheromatous or covered with villous vegetations. There is always danger of rupture of these aneurisms; the more so if there be present hypertrophy of the unaffected portions of the heart.

Sometimes the papillary muscles are especially affected by this chronic inflammatory action, which is degenerative in character. In these cases there is auriculo-ventricular regurgitation from imperfect closure of the valves; the chordæ tendineæ and papillary muscles become elongated from the softening process, and so permit the valve flaps to wash backwards on the ventricular systole and produce a murmur.

Localized myocarditis, from an embolon finding its way into a branch of the coronary arteries, as may occur after suppuration or destruction of the lungs, leads to the formation of an abscess; then there is molecular necrosis and the formation of pus. This abscess may perforate the endocardium and pour its contents into the circulation, or it may take the direction of the pericardium, and form a "false" aneurism of the heart. This is liable to rupture and so to cause death.

The symptoms of myocarditis, when general, are probably those given by Hope in the case related above. Where the mischief is localized, as in cases where scars are found after death, the symptoms must be very obscure, and the mischief in the heart-wall rarely be suspected even. Probably there are dyspnœa, embarrassment of the heart's action, feeble pulse, with irregular cardiac action, and a tendency to syncope. Piorry thought that there was a peculiar pain in myocarditis which was aggravated on each systole. The difficulties in the way of such a rapidly changing pain being recognized by the patient are insuperable, and "the pain of Piorry" (which he thought pathognomonic of myocarditis) has been relegated to the list of abandoned diagnostic minutiæ.

Where there is aneurism of the apex of the heart, Skoda has thought it possible to detect a bulging in the intercostal parietes, and he is entitled to credence from the accuracy of his observations. Where there is the stenosis of Dittrich, a systolic murmur may be heard at the base, while, when the papillary muscles

are elongated, a systolic murmur at the apex, and usually the left apex, will be audible.

As to the prognosis and termination of chronic myocarditis, much will depend on the area affected. The scars of Dittrich show that repair is not impossible when the mischief is very localized. But where aneurism, "true" or "false," is formed, the danger of sudden death from rupture of the sac must be very great. The difficulty of diagnosing such a case in life must be great, almost insuperably great. Where the softening is general, the fatal termination can never be far distant.

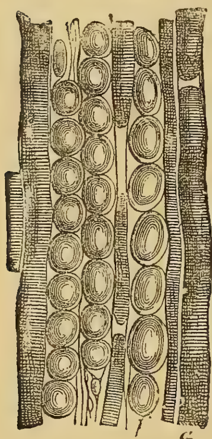
Chronic myocarditis may lead to the development of connective tissue of variable character, producing false hypertrophy, or, at other times, induration of the heart, or, as Schrœtter holds, perhaps even fatty degeneration, or, at other times, fatty infiltration. But it seems desirable to discuss these different pathological conditions separately, as distinct diseases.

Fatty Infiltration.

Fatty infiltration of the heart is a disease distinct from fatty degeneration. In fatty degeneration the muscular fibrillæ are themselves the seat of the morbid change—a histolytic process. Fatty infiltration of the heart is a development of adipose tissue betwixt the muscular fibrillæ. The connective tissue lying amidst and between the muscular fibres, undergoes the same changes that are found in ordinary adipose tissue. "Histological inquiries teach us that, when an animal is fattening, the minute drops or specks of fat normally present in certain connective-tissue corpuscles are seen to increase in number, the protoplasm enlarging at the same time. As these specks increase they coalesce into drops, which by similar coalescence form larger drops, until, the protoplasm, first ceasing to increase and then diminishing, the original connective-tissue corpuscle is transformed into a fat-cell, with a remnant only of protoplasm gathered round the nucleus, and forming an imperfect envelope round the enlarged contents" (M. Foster). Fatty infiltration of the heart is, then, a disease of obesity, which fatty degeneration is not,—for it is commonly found with leanness, and even with emaciation.

The following illustration will convey a clear impression of the pathological change known as fatty infiltration:

FIG. 35.



Fatty Infiltration of Heart.—(From Rindfleisch.)

From it the development of fat-cells will be readily caught, and also, what is more important, the effects of this growth of adipose tissue upon the neighboring muscular fibrillæ. It is this last which gives to fatty infiltration its sinister aspect. Mere accumulation of fat upon the heart may impede its movements, and by filling up the space within which the heart can expand in the firmly walled thorax, prevent to some extent the heart's distension in diastole, but beyond that it is harmless. But when the intermuscular connective tissue is converted into fat-cells, pressure is exercised upon the neighboring fibrillæ, which become more or less wasted and obliterated. Often the outer layers of muscular fibrillæ become destroyed and their place taken by these fat-cells, while the inner layers are histologically sound. But this reduction of the thickness of the muscular wall enfeebles the heart, and so causes the evidences of cardiac adynamy. It would appear from different authorities, that rupture of the heart is very rarely met with in heart-walls so attenuated.

The symptoms of fatty infiltration are obscure, as are those of heart failure. Hope says, "Its signs, so far as I can yet judge, are: 1, diminution of the sounds, especially the first; 2, irregular pulse, without valvular disease; 3, 'oppression,' retarded circulation, producing cerebral, hepatic, and other congestions." The signs and symptoms are those of a dilated or weakened heart, but if found along with obesity, especially comparatively early in life, such symptoms and signs will indicate with considerable certainty fatty infiltration of the heart. Physical examination throws little light on such a case. There is no visible impulse, the apex beat cannot be felt, partly owing to its feebleness, partly to the thick fat chest parietes, while, on account of the latter, percussion can give no exact impression of the area

of cardiac dulness, and if it did, could not determine whether the increase was due to dilatation or deposit of fat. The diagnosis really is a question of probability founded on certain data, which, however, are insufficient for a definite diagnosis.

Of course the prognosis of such cases is bad, and the direction downward. As to any treatment to be adopted, it must be such as would reduce the general obesity without, however, being too weakening, and so impairing the muscular fibre of the heart remaining structurally sound.

Fatty Degeneration.

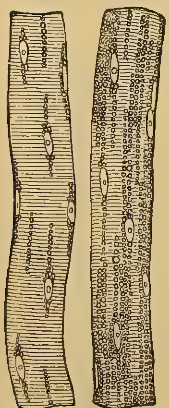
Fatty degeneration of the heart is a disease of the most serious character. It is a necrobiotic process affecting the muscular fibrillæ—a species, indeed, of death in life; for certain muscular fibres practically die within the living organism. It is the same change in many respects as that of adipocere, where a piece of muscle is converted into a greasy mass, either by placing it in running water, or in a highly diluted mixture of nitric acid and water. The muscular mass becomes paler, softer, less consistent, and, ultimately, is converted into a piece of dirty white fat, all structural formation being entirely lost. Wagner found that similar changes were produced when albuminous substances were inclosed in the abdominal cavity. It is a true histolysis of muscular fibre, a retrograde tissue metamorphosis by which albuminous substances are resolved into their more primitive elements—hydrocarbons and ammonia.

Such a process is a form of molecular death; but, nevertheless, is not always a morbid process. It is the normal process by which the enlarged uterus, after delivery, is reduced to its normal unimpregnated size. Neither can we say that, when not a physiological process, as in the involution of the uterus, it is necessarily incompatible with repair. It occurs in palsied muscles, as in the essential paralysis of infants; it is found in muscles put at rest for the repair of a broken bone. When the muscle is not put in action by its motor nerve, it would seem that instead of normal removal of waste or effete material, and the laying down of new normal histological elements, the waste accumulates, the

muscle shrinks;* but when movement is once more instituted, then the waste is removed, and the fibrillæ recover their normal structure. It is important to bear these facts in mind, as they bear on the momentous question,—is fatty degeneration of the heart always a hopeless condition?

Fatty degeneration is the non-renewal of the muscular fibre from the protoplasm, or germinal matter; and, as the following plate will show, the molecular necrosis commences first in the neighborhood of the protoplasmic mass, and extends, therefrom, throughout the muscular fibre. As to the visible changes so produced, they are distinct. To the naked eye the color of the muscular fibre is changed from red to a dead-leaf hue, or a dirty yellow; not alike throughout the heart, but more pronounced in some parts than in others, giving the heart a mottled appearance. Under the microscope, according to Dr. Ormerod, whose description has not been excelled by more recent writers, in the first stage the transverse striæ are less distinctly marked than in normal heart fibre. The fibrillæ present a singularly granular appearance. "On opening a heart thus affected, the interior of the ventricles appears to be mottled over with buff-colored spots of a singular zigzag form. The same may be noticed beneath the pericardium also; and in extreme cases, the same appearance is found on section to pervade the whole thickness of the walls of the ventricle and of the carneæ columnæ. Of these latter, the musculi papillares seem most liable to be affected: not to say that this form of disease never occurs in the walls of the auricles—at least, I have never seen it there.

Fig. 36.



Fatty Degeneration of Heart Fibre, showing more and less advanced stages. (After Rindfleisch.)

"Microscopic examination reveals the nature of these spots; they are not deposits, but distinctly degenerated muscular fibres. The outline, not merely of the masses but of each single fibril, is accurately preserved. Instead, however, of transverse striæ and nuclei, the evidences of active vitality, there

* Rokitansky states: "We may then find in a limb nothing of its muscles, but remnants of tendon and aponeuroses, with their prolongations inwards."

is little to be seen but a congeries of oil-globules. The whole history of the degeneration may be traced in one of these little spots. First, from the immediate neighborhood of the spot we may obtain healthy muscular fibre; then the transverse striæ become less distinct, they are rows of dots rather than continuous lines; then the intervals between the dots become wider, and the dots themselves run into longitudinal rather than transverse lines; and then all regularity is lost, and the dots appear to stud the surface all over, like the points on a bit of fish skin. Probably long before this time the fibre has lost all its properties as a muscle; but there are further changes to observe; for now, mixed with these minute dots, are to be seen small oil-globules, which increase and coalesce till the fibril presents little else but a congeries of oil-drops contained within the sarcolemma.

"This is not the only change which the fibres undergo; for with whatever care they are disintegrated, they are found to be short, and as if unusually brittle, a general condition which may, perhaps, be of more serious importance than the actual fatty degeneration."

The granular matter, the first minute speck of fat, has been found both by Paget and Rindfleisch to appear round the nucleus. Von Dusch says, that the dark dots which mark the earliest stages consist of the coloring matter of the muscular fibre, and are not unlike the pigment granules of brown atrophy. This gathering together of the pigment matter producing a lighter shade of muscle, is analogous to the change of hue in the color of the frog's skin by the diffusion of (darker shade), and gathering together (lighter hue) of the pigment matter in its skin. The necrobiotic change does not spread evenly throughout the mass of the cardiac muscle, but is found more marked in some fibrillæ than in others regionally near them; and in some the cross markings are only blurred, when in other neighboring fibrillæ the sarcolemma contains but a row of oil-drops amid debris. As to the spread of fatty degeneration throughout the heart, Hayden says, "Where fatty degeneration is due to obstruction of minute branches of the coronary arteries, its area is often very limited. It not unfrequently also appears upon the inner or outer surface of the heart, in the form of light fawn-colored spots, varying in size from the diameter of a pin's head to that of a split pea."

As to the coexistence of fatty degeneration with fatty infiltration, Rokitansky says, "There is a form of degeneration to which the muscular structure of the heart is liable, particularly when hypertrophied; it is met with, also, but not so frequently, in the muscular coats of other organs when they are hypertrophied, but is very seldom seen in the muscles of animal life. It is characterized by the development of minute particles of free fat between the primitive muscular fibres. At the same time the striated sheath of the fibre disappears, and the muscle changes to a dirty yellow or fawn color and becomes friable." Hayden says, "Whilst, in the majority of cases, superficial accumulation of fat upon the heart coexists with fatty degeneration of its substance, interstitial or interfibrous deposition of fat is rarely met with in conjunction with fatty degeneration."

During the process of fatty degeneration there are certain naked-eye changes instituted. The color is paler, for the reasons given above, and the consistence is altered. The tissue is friable, and tears with a sort of fracture, and crumbles down readily under the finger and thumb. In advanced cases the finger is easily pushed through the ventricular wall, as it may be through several thicknesses of wetted paper. It feels greasy and unlike normal heart-wall, and a warm knife passed through it looks oily. There is not necessarily any change in bulk, nor does the degenerate structure inevitably take on dilatation. The secondary dilatation is the rule where hypertrophy has existed previous to the degenerative changes. From the decay not being evenly spread throughout the heart, a mottled appearance is found. The pale buff color of the heart contrasts with the large blue prominent coronary veins, which are usually tortuous and atheromatous. Nor is there necessarily a deposit of fat along the sulcus in which the veins lie, but when it is present it makes the contrast in color all the more striking. The coronary arteries are not uncommonly tortuous, rigid, and atheromatous. The heart may be of normal size, or it may be hypertrophied and dilated; the latter being more common, as we shall soon see. Older writers speculated as to the readiness of the new fibres in hypertrophy of the heart to undergo degenerative change; but of this there is no proof. Why fatty degenerations should follow hypertrophy will soon be apparent.

Causation.—The causes of fatty degeneration of the muscular

fibrillæ of the heart are various. It is found in phosphorus poisoning; where the voluntary muscles, the liver, spleen, and kidneys are also the subject of this necrobiotic change. It is also found with poisoning by various acids; and Von Dusch gives a list of these acids, which comprises sulphuric, nitric, phosphoric, oxalic and tartaric, where this change has followed poisonous doses. What is the action of these acids upon albuminous tissues is not yet known, but apparently they possess a power to split them up. Possibly it may be through their affinity for the ammonia of the albuminoids, leaving the hydrocarbons behind in the form of fat.

Virchow has noted the presence of fatty degeneration of the heart in chlorosis; and Prof. Ponfick has described a series of cases of what he terms "anæmic" fatty degeneration. Here there is a deficiency of red blood-corpuscles, and of the fibrin in the blood, with an excess of white corpuscles. There is also some fatty change in the inner and middle coats of the arteries. (By the courtesy of the late Prof. Traube, I was shown one of these cases, in life, in the wards of La Charité, Berlin. The woman, for in women it chiefly occurs, was pale and waxy-looking. The breathing was shallow; the pulse fast and very feeble, and the heart's sounds and impulse were scarcely to be detected, though the chest-walls were thin, and the body generally wasted. The general features of the case were those of pernicious anæmia; and the heart symptoms were by no means prominent.) Quain has observed fatty degeneration of the heart in young persons associated with recurrent hæmorrhage. Probably in these cases the number of red corpuscles is so diminished that proper oxidation of effete tissue is impossible; and the heart, the most active of all muscles, is the seat of the most marked changes due to the non-removal of fatty debris. It is possible, too, that assimilation is defective, that new tissue could not be laid down if the fatty debris were removed.

Such are the associations of fatty degeneration of the heart with anæmia in comparatively young persons.

The most common form of fatty degeneration of the heart is that found in advanced life, for which the name "senile" has been proposed. This form is associated with atheromatous arteries, and with failing hypertrophy. Another form also found

in advanced life chiefly, is not connected with any enlargement of the heart, either pre-existing or concomitant; this form will be described later on. "Senile" fatty degeneration may be applied to both forms of the disease; but the term "fatty degeneration of failing hypertrophy" is that which seems to me to convey the most vivid conception of the associations of the first form of this necrobiotic process.

It was once supposed that the new fibres of hypertrophy were more liable to be the subject of fatty change than the normal fibres of the heart. Of this there is no evidence whatever. Nevertheless, there is equally no doubt that a hypertrophied heart is more liable to become ultimately the subject of fatty degeneration than a heart of normal size. Nor is it difficult to comprehend the why of this. One of the commonest causes of heart hypertrophy is a high blood-pressure in the arteries; and this is most frequently seen in those who are the subjects of renal disease. As the processes of heart change in their different stages will be given in the chapter on "The Gouty Heart," it is unnecessary to give any lengthened account of them here. It is sufficient to start from the point of hypertrophy of the left ventricle, with commencing atheroma of the aorta and large arteries. As the atheromatous process progresses the elasticity of the arteries becomes gradually diminished; and their recoil, after distension by the ventricular systole, is much impaired. This arterial, or perhaps rather aortic recoil, is the power which propels the blood into the coronary arteries. The blood driven backwards, as well as forwards, by the aortic rebound, is arrested in its backward flow by the aortic valves, and so eddies powerfully in the sinuses of Valsalva, from whence spring the nutrient arteries of the heart. When the elasticity of the aorta is impaired, the pressure of the blood in the coronary arteries is lessened, and the blood supply to the heart is diminished. At the same time that the recoil of the aorta is being impaired, the coronary vessels are themselves the subject of the atheromatous process. As will be seen when the chapter on "Atheroma of the Arteries" is reached, overdistension is the chief exciting cause of the arterial change. The coronary vessels placed at the base of the aortic column are subjected to the full unbroken force of the aortic rebound, and so

are more violently distended than any other arteries of the same diameter. Consequently we commonly find the coronary vessels more atheromatous, more extensively diseased, than the rest of the arterial system. This atheromatous process in the coronary arteries elongates them, and so causes them to become tortuous; and by their tortuosity the blood-flow in them is impeded. Atheroma being a disease of the tunica intima, the growth diminishes the calibre of the arteries, lessening their lumen; and so reducing the blood supply to the muscular wall of the heart. Thus we have (1) diminished aortic rebound; (2) impeded blood-flow; and (3) arteries of lessened diameter; and consequently (4) impaired nutrition of the heart. This is felt, of course, most where growth is active; and Rindfleisch has pointed out that fatty degeneration commences from the germinal spots—the masses of protoplasm.

This mural decay is not equally spread throughout the heart; it is most common in the left ventricle, which is also most commonly the seat of the hypertrophy. Neither is the heart-wall uniformly diseased alike all over. Fatty degeneration is more pronounced at some spots than others (Ormerod), and we are compelled to suppose that this is due to changes in the branches of the coronary arteries. Some twigs become more diseased than others, and so the portions of muscular fibre supplied by them are more diseased than those portions where the arterial twigs are less affected and the tissue nutrition less impaired. "Where fatty degeneration is due to obstruction of minute branches of the coronary arteries, its area is often very limited" (Hayden). The causes which affect the heart generally, and especially the diminution of the calibre of arteries by atheromatous changes, affect localized areas still more, and as some arterial twigs are more diseased than others, so the tissues supplied by them are found in a state of more advanced histological decay than the muscular fibres fed by arterial twigs less diseased. The mottling of unequal decay is readily comprehended.

The hypertrophied heart, no longer sufficiently supplied with blood, commences to dilate (Niemeyer) as the mural decay spreads. It can no longer successfully resist the dilating action of the contained blood, and yields to this distending force. The term, now in common use, to describe this condition is "failing hypertrophy."

The symptoms and physical signs of this complex condition are rather confusing, and, without a thorough comprehension of the morbid anatomy of the disease, are scarcely likely to be read aright. The blending of the power of hypertrophy with the feebleness of the diseased portions of the heart-walls, produces a complexity of symptoms which requires some thought for their correct interpretation, and correct interpretation of them is of the utmost importance for an accurate prognosis, in which many interests may be involved.

Such is the common form of fatty degeneration of the muscular fibrillæ of the heart, and there are some considerations connected therewith which may as well be reviewed at this as at any other point. In his address on medicine at the meeting of the Brit. Med. Assoc. at Leeds, in 1869, Sir W. Jenner spoke of fatty degeneration of the heart as "a preservative lesion." He said that when it was first so spoken of in the Pathological Society of London, the announcement was received with shouts of derisive laughter. But as knowledge progressed the statement was found to be substantially correct, and the society, who once so laughed, is now at one with the author (Edmunds Crisp) of that remarkable statement. As the arteries become atheromatous and readily ruptured, an hypertrophied heart contracting with unimpaired vigor would be a source of great danger, and arterial rupture would be more common than it is. A fair balance of power betwixt a decaying heart and degenerate vessels, gives a better prospect of life to the patient than would be the case if the rotten arteries were still subjected to the shock of the systole of an hypertrophied ventricle, with walls structurally sound.

Apoplexy is the danger, *par excellence*, of the earlier stages of heart hypertrophy, with arteries undergoing atheromatous changes. The increased blood-pressure in the arteries, maintained by cardiac hypertrophy, tests the arterial coats severely; and rupture at some spot in a more advanced condition of decay than the bulk of the arterial walls, is a common occurrence. As the heart-walls are undermined by this necrobiotic process the risk of apoplexy is greatly diminished; but a new danger is substituted for it. Failure of the heart in diastole is the danger to be dreaded in the later stages; and the patient, having survived the risks of apo-

plexy, drifts into the graver peril of heart failure from fatty degeneration.

Such then is the history of a morbid process of the most enthralling interest. Families of the gouty, or arthritic diathesis, are the subjects of the changes just detailed. Death from apoplexy, from the massive heart bursting the rigid, atheromatous arteries is the danger which threatens such individuals in the fulness of their strength and the enjoyment of robust health. Angina pectoris is found both in the earlier and later stages; but is comparatively devoid of danger during the earlier stages. But as time goes on the risk of arterial rupture wanes; and where the members of the family, who have died in middle age, died from apoplexy, or were the subjects of paralysis, those who survive to a more advanced age die suddenly from heart stoppage, or are the subjects of dropsy and the other outcomes of heart failure. The preservative lesion has undoubtedly protracted their existence; but it, in its turn, constitutes a graver and more certain danger. To reflect on this long and extended morbid process; to watch hypertrophy yielding to fatty degeneration; to note how the indications of the one are merged in the advancing evidences of the oncome of the other, forms a subject of the greatest clinical interest.

As the power of the heart wanes, the aorta is less perfectly filled on each ventricular contraction; the rebound of the arteries is in direct proportion to their distension, and the blood supply to the coronary arteries becomes less and less sufficient, as the necrobiotic process spreads. The tendency is steadily downwards; and the therapeutic art exercises but little control over the morbid changes of failing hypertrophy. The great aim is to reduce the demands upon the failing heart; to supply a blood as rich in nutrient material as is possible, and to act upon those portions of the heart remaining comparatively sound, when stoppage in diastole is impending.

Fatty degeneration of the heart is found under other circumstances, where it is certainly a "senile" change in the truest sense of the word. As this form has not been so carefully described as the one given above, a somewhat large space must be devoted to its pathology, involving several quotations. In speaking of senile

changes, Michael Foster writes: "Two changes, characteristic of old age, are the so-called calcareous and fatty degenerations. These are seen in a completely typical form in cartilage, as, for instance, in the ribs; here the protoplasm of the cartilage corpuscles becomes hardly more than an envelope of fat-globules, and the supple matrix is rendered rigid with amorphous deposits of calcic phosphates and carbonates, which are, at the same time, the signs of past and the cause of future nutritive decline. And what is obvious in the case of cartilage is more or less evident in other tissues. Everywhere we see a disposition on the part of protoplasm to fall back on the easier task of forming fat rather than to carry on the more arduous duty of manufacturing new material like itself. Everywhere, almost, we see a tendency to the replacement of a structured matrix by a deposit of amorphous material. In no part of the system is this more evident than in the arteries; one common feature of old age is the conversion by such change of the supple elastic tubes into rigid channels, whereby the supply to the various tissues of nutritive material is rendered increasingly more difficult, and their intrinsic decay proportionally hurried." Such, then, are the senile changes by which fat is deposited in lieu of normal tissue. In speaking of fatty degeneration of the heart, Stokes writes: "In its higher degrees of development this affection is most frequently met with in persons who have passed the prime of life; but minor shades of it occur in young persons, especially where there is a complication with other visceral diseases, as for example, pulmonary tubercle. On the other hand, some of the most remarkable instances are found in very old and long bedridden subjects; and it is observed that in such cases the alteration is not confined to the heart, but extends also to the voluntary muscles, and even to the skeleton, producing atrophy and fragility of the bones, with a great deposit of oily matter in the cavities and cancelli of the osseous tissue. Though varying and apparently opposite, its exciting causes are generally reducible to those which would induce a depraved hæmatosis. The overfed and luxurious on the one hand, and the victim of want on the other, are liable to the disease." This form of extensive fatty metamorphosis of tissues is thus spoken of by Rokitansky: "The fatty degeneration which is brought on by a seden-

tary and luxurious mode of living, and by spirit-drinking is, for the most part, accompanied by a great development of fat throughout the system; the liver usually contains a quantity of tallowy substance, the heart is loaded with fat, and its muscular tissue is more or less metamorphosed into the same substance;* moreover, in old people more particularly, the medulla of the bones connected with the altered muscles is in excess, and the bones are in a state of osteoporosis or eccentric atrophy, and are easily broken. The fat by which the muscular tissue is supplanted varies in its character. In some instances it resembles ordinary healthy fat; sometimes, especially in persons advanced in years, it is of a dark-yellow color, loose and diffluent, and sometimes in its consistence and whiteness it is remarkably like mutton suet. It assumes this last character particularly when the change results from anchylosis." There would appear to be a general tissue decay throughout the body, in some cases more conspicuous in one part or tissue than in others. Basham (on dropsy) in speaking of dropsy connected with the heart and lungs, writes: "Here, therefore, as in renal dropsy, we recognize, not a local, but a general disorder; a determination of cell-development not confined to one texture or organ, but widespread, and signifying to us in studying the pathology of these disorders that it is not to the organ which gives the most prominent indication of disturbance that our examination should be limited; but if we are to render available the results of our observations for the purpose of treatment, we must take into consideration the evidence which has been here offered, that in these dropsical diseases we have a decaying vitality—a decreasing power of elaborating, or forming out of the elements of food, cells fitted for, and equal to, the performance of their several functions."

This general widespread tissue decay is obviously a senile change, but at times the physiological age is far in excess of the chronological age. In some organisms the tissues grow old prematurely, and senile changes are found in comparatively young persons. The term "Climacteric Decay" has been given to this general and often premature decay. Sir Henry Halford described it minutely. The abstract of his essay, made by Cop-

* Rokitansky constantly speaks of fatty accumulation in the heart as found along with fatty degeneration of the muscular fibrillæ.

land, is as follows: "It usually comes on insensibly. The patient first complains of fatigue upon slight exertion; his appetite becomes impaired; his nights are disturbed or sleepless, and his mornings unrefreshed. The tongue is somewhat white; the pulse a little accelerated; the face attenuated, occasionally slightly bloated; the body emaciated, and the ankles and legs disposed to swell. The urine is not deficient, but the bowels are sluggish, and pains, with vertigo, are occasionally felt shooting through the head and various parts of the body, but are not possessed of the rheumatic character. As the vital exhaustion proceeds, the stomach loses all its powers; the emaciation is greater; the lower limbs more œdematous; restlessness through the day and sleeplessness through the night increase, and all the vital manifestations, mental and physical, are gradually extinguished. Such is the usual progress of the simple form of the disease, or rather gradual decay of the vital energies—a decay which is not peculiar to, but which may occur at any time intermediate between the grand climacteric periods. This simple form of decay is, however, less frequently observed than its complication with other affections. Persons who, together with the anxieties, griefs, and distresses of life, have been subject to disease of some particular organ, as of the lungs, liver, brain, heart, etc.; who are of a gouty, rheumatic, or calculous diathesis, generally experience, at these epochs,* an aggravation of such diseases, which assume a more dangerous character from the vital decay which is thus attendant upon them."

These excerpta from various high authorities will probably be more convincing to the reader than a succinct description of the vital decay found in some persons, of which fatty degeneration of the heart is a prominent factor. The degenerative changes are not confined to the heart, but are found in the lungs, liver, kidneys, and viscera, and the morbid process goes on in all the organs alike, if not at an equal rate in all. They do not stand to each other in the relation of cause and effect, but are rather the results of some general condition common to, and standing to each in a causal relationship. As to the prognosis and treatment

* The sixty-third and eighty-first years were the "grand climacterics;" the lesser ones were the forty-ninth and twenty-first years.

of this condition, the first is as bad as it well can be, and the latter is only of slight palliative value in most cases.

On the other hand, there are cases of general impairment, commonly with an alcoholic factor present, with great enfeeblement of the heart, where an encouraging result is sometimes attained by albuminoid food, plenty of fresh air, and tonics and chalybeates. We are not yet in a position to affirm that in every such case the prospect is hopeless. There are grounds for believing that in some cases the tendency of the tissues to split up into fat, or for protoplasm to make fat instead of normal tissue, may be got over, and a restoration of normal nutrition be attained. The late Warburton Begbie taught that in some cases fatty degeneration of the heart was amenable to treatment. From the above considerations we can see that this fatty degeneration is a histolytic process, variously instituted. That it commences in the germinal matter and spreads therefrom into the formed material of the muscular fibrillæ, gives us the impression that it is an arrest or perversion of nutritive processes, seen first where the nutritive processes are most active,—namely, in the neighborhood of the protoplasmic masses, from whence it spreads through the formed material, as that wears out in the performance of its function, and is no longer perfectly renewed.

As to fatty degeneration of the heart, unaccompanied by evidence of pre-existing hypertrophy or of general decay, it must be rare, and when found we are compelled to suppose that the nutrition of the heart is impaired by local causes of malnutrition, as by an atheromatous mass at the orifice of a coronary artery, by an atheromatous and earthy scale forming so as to occlude a coronary vessel, or by adhesion of the pericardium along the track of the coronary vessels, for the sulci in which these run are not always deep enough to protect them from this danger. But these are the rarer and more obscure forms of fatty degeneration of the heart.

As to the age at which this affection is most apt to develop, it is certainly that of advanced life; the greatest number of deaths from fatty degeneration occurring in any one year is stated by Sir Thomas Watson to be the sixty-third (the first grand climacteric). But probably it is found rather in advanced life than

any particular year or period. Hayden has collected eighty-eight cases, from which it would appear that fatty degeneration of the heart is most prevalent between the ages of sixty and seventy years; and next between forty and fifty. As to the sex in which it occurs it is comparatively a disease of men, both in the form which accompanies failing hypertrophy, and that which is more truly senile. But a condition of debility of the heart, where the symptoms of fatty degeneration of the heart are closely simulated, is often found in women at or about the menopause.

As to the effect of occupation in producing this necrobiotic change, little can be said. In my own experience it has been frequently seen in elderly publicans. It is said to be common among London gin-drinkers; but that merely means that it is found with want, with the absence of proper food, and with general constitutional decay. Chronic alcoholism, and the arterial degeneration which is produced thereby, especially when syphilis is also present, are, no doubt, associated with this necrobiotic change. The general premature wornout state found in drunkards is favorable to the development of fatty heart; but it may be brought about under other circumstances. Probably a sedentary existence, with the indulgence in rich food, is conducive to such a disease. It is said to be a frequent cause of death amidst judges. The non-removal of waste matter in the tissues must conduce to it; and where the respired oxygen is met in the body by the presence of large amounts of hydrocarbons, the removal of waste albuminoid tissues must be interfered with. A sedentary life with a generous dietary will favor fatty degeneration of the heart (Fettherz), as well as fatty deposit on the heart (Neufettbildung). But obesity has nothing to do with fatty degeneration of the muscular fibres of the heart; nor is extreme emaciation any protection against this necrobiotic process. Stout persons and lean ones alike die of fatty degeneration of the heart; and it may be found in every rank and position of life, from the millionaire to the bricklayer's laborer.

The *physical signs* of this degenerative decay are negative rather than positive, and the absence of action and power are the chief indications. To inspection little is revealed except the absence of impulse; even in comparatively thin persons, and

when placed so that the light shall fall clearly on the chest-walls. To palpation the impulse is faint or totally wanting in those cases where there is no increase in the size of the heart. In failing hypertrophy there is a tumbling, rolling, irregular action of the heart, very similar to that of a weak dilated heart; with a sensation of diffusion as if the apex were broadened. But these are the remains of the positive signs of hypertrophy, rather than the indications of fatty degeneration. The concomitants—as the age of the patient, and the advanced condition of the atheromatous arteries, will help to distinguish the irregular action of the heart in failing hypertrophy, from the similar action in dilated heart and in mitral insufficiency.

Probably percussion yields very little positive evidence as to the state of the heart. In cases of atrophy the area of cardiac dulness may be lessened, but not to any very perceptible extent. In failing hypertrophy, the ossification of the costal cartilages, and the presence of emphysema of the anterior lobes of the lungs, prevent anything like accuracy being possible. Error is easy under such circumstances. (See a case of Clifford Allbutt's, given in the following chapter.)

Auscultation furnishes us with the most important information derived from the physical signs. The alteration in the first sound is marked, especially by contrast with the thin, clear, second sound of the aortic valves. Indeed, not uncommonly the second sound remains still accentuated in failing hypertrophy. Often the first sound consists solely of the clear flapping together of the auriculo-ventricular valves, and is clear and thin, resembling the second sound in character. The muscular portion of the first sound is absent or much impaired. This loss of or alteration in the first sound, taken along with the evidences of arterial degeneration and osseous alteration, will, generally, make the diagnosis fairly clear in failing hypertrophy. Hope, in the last edition of his book, refers to the difficulty he once experienced in detecting softening when complicated with hypertrophy. He says the first sound in hypertrophous softening is of a short, flapping character, while there remain the ordinary physical signs of hypertrophy—the augmented impulse, either constantly or with occasional beats. The irregularity of the pulse is an additional indication of soften-

ing, because this sign is foreign to mere hypertrophy (4th edition, p. 417). In the early stages these will be rather the indications of hypertrophy, with commencing irregularity in the pulse, the irregularity telling of mural decay, the strong impulses of a fair amount of heart-tissue remaining sound. To judge of the relative amount of fatty degeneration, and of muscular fibre remaining structurally sound in a case of failing hypertrophy, repeated examination is necessary, and even then allowances must be made for temporary improvement and appropriate treatment. In elderly persons, when not well, the heart is apt to become very feeble for a time, so that fatty degeneration must be gravely suspected, and yet, with improvement in the general condition, there follows a restoration of the normal sounds of the heart and fair functional vigor. The action of the heart, which is the subject of fatty infiltration in the interfibrillar areolar tissue, with wasting of the fibrillæ from pressure upon them; or of the heart, which is laden with fat under the pericardium, resembles closely, alike in objective and subjective indications, that of the heart which is undergoing fatty degeneration.

Walshe says of fatty degeneration of the heart, "The physical signs are those of a soft heart. Weak impulse, indistinctness of the apex beat, unchanged percussion dulness (unless there be alteration of the bulk from some other cause); a feeble, toneless, short first sound; a long first silence, and a feeble second sound. (This may be of better tone at the second left than at the second right cartilage, if the fatty disorganization be, as it often is, in great excess in the left ventricle.) The pulse is irregular in force and rhythm, either constantly or from time to time, under excitement, the influence of flatulence, indigestion, effort, etc. On such occasions it may become exceedingly frequent. I have known it uncountable, in the main, from frequency; in part, however, from irregularity. Infrequency of pulse, occasionally met with, is, in some cases, referable to the weakness of occasional systoles; but the systoles are themselves sometimes much less frequent than natural."

With this may be contrasted the physical signs of fatty accumulation under the pericardium, which produces minor annoyances and some physical signs—sketched by the same master-

hand. They consist of "Sensation of oppression, or even pain, about the pericardial region; syncopal feelings on exertion; inability to walk quickly on level ground and to get up hill, except with great and painful effort; inclination to coldness of the extremities; feeble (but, as far as I positively know, regular) pulse; sluggish action of the liver and bowels; occasional giddiness and feeble cardiac impulse, with a too extensive dulness on percussion; the sounds, especially the first, being weak and toneless. These were the symptoms and signs in the only person (a male, ætat 64) I happen to have observed during life and opened after death, whose heart was at once loaded with subpericardial fat, and positively free from softening or intra-sarcolemmous oil."

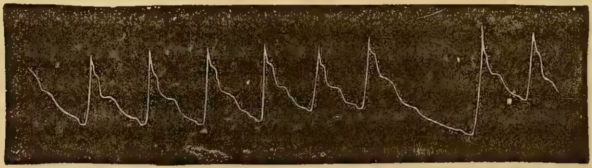
It seems that the indications of cardiac enfeeblement from fatty accumulation are very much allied to those of fatty degeneration, which are, according to Hayden, as follows: "There is in all cases evidence of partial failure of the circulation, under the form of weak, irregular, intermittent, or very slow action of the heart and radial pulse; pericardial oppression or pain of an intermittent character, the latter frequently extending down the left arm as far as the elbow; palpitation on making any unusual effort, physical or mental; inability to resist the operation of heat or cold, or preserve the thermal equilibrium under extremes of temperature, pallor of surface; readiness to perspire, and recurrent syncope." He continues: "The symptoms indicative of a fatty condition of the heart are individually of little value, but combined in groups they assume an affirmative significance."

But beyond the mere physical signs, there are a number of objective phenomena connected with fatty degeneration of the heart of the most important character. They may be connected with the vascular system, or with the nervous system, or be discernible to the eye.

Palpitation may be present, but it is not indicative of fatty degeneration; it tells that the heart is readily embarrassed, but beyond that has no diagnostic value. It is rather found where there is failing hypertrophy than where the heart is of normal size. It is an active evidence of inability in the heart-walls; instead of it, in degenerate walls, there is a tendency to syncope with vertigo. Palpitation is the characteristic of dilatation; syn-

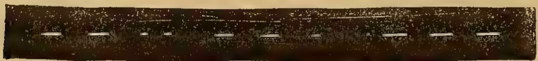
cope of structural degeneration. Palpitation is connected with the dilatation of the fibres remaining sound, not with those that are converted into fat-globules. Irregularity of action is not uncommonly found. It is readily produced by effort in hearts commencing to decay, which beat rhythmically when the organism is at rest. In more advanced cases there is persisting irregularity, with palpitation superimposed on slight effort, as mounting a flight of stairs, straining at stool, or by any cause of mental perturbation. As the case progresses, the ominous halt of intermittency is developed along with marked irregularity. But intermittency varies considerably. With some elderly persons with corded arteries and a circulation readily embarrassed, and yet in fair health, if quiet is maintained the intermittency is rhythmical, and occurs after an interval of so many beats with much regularity. The pulse is as follows:

Such a pulse is found with many elderly persons, who live on for years, and is not of grave omen, comparatively, at least.



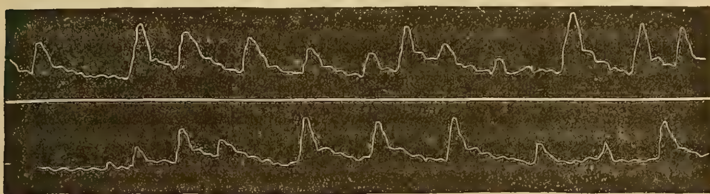
Personal observation enables this statement to be made with considerable confidence. But when the intermittent halt occurs amidst a number of irregular beats, then the aspect of the case is much worse.

The following form of pulse is indicative of a very grave state.



indeed. Sudden death may occur at any time, and life is never

far prolonged, especially when the intermittent halt is rendered more frequent by slight exertion. The long diastolic pause,



sooner or later, is no longer followed by a systole, and death is the result. It may occur in bed, but more commonly is the result of effort, as hurrying to catch a train, or pursuit of an omnibus. Personally, I am inclined to think that some effort during the day usually precedes sudden death in sleep. It is well in cases of advanced disease of the heart walls to compare the radial pulse with the beats of the heart. By this means true diastolic pause can be distinguished from the pause in the pulse due to a ventricular contraction, too feeble to cause a pulsation in the radial artery. The halt amidst the flutter of irregularity is more ominous than when it occurs amidst normal beats. When with the forms of pulse just described the first sound is short, and rather valvular than muscular, structural disease of the heart is almost certainly present. "Slow pulse, that is under sixty in the minute, is very rare. It is worthy of remark, that in most of the reported cases of slow pulse in which a careful dissection has been made, the coronary arteries were extensively diseased, and the substance of the brain soft and anæmic; whilst, on the other hand, either of these lesions singly may be commonly met with, unassociated with slow pulse" (Hayden).

As to that form of irregularity of pulse where a number of beats occur at a certain rate, say eighty, and then a number at ninety-six per minute, the irregularity is caused by the effects of respiration and a varying pressure of the elastic lungs. The irregularity of pulse found in emphysema is due to pressure change (Broadbent). And the long interval betwixt the first and second sound, which gives the heart sounds a "tick-tack" character, are

not associated with fatty heart, but with a dilated heart with hardening of the walls (Broadbent). B. W. Richardson holds that cardiac intermittency is a purely nervous phenomenon; but most authorities will admit that it is found usually in cases where there are evidences of structural disease.

Probably the cardiac ganglia are involved in the same malnutrition as the muscular fibrillæ, and then the rhythmic discharge is momentarily arrested. At other times the discharge excites so feeble a contraction that its impulse is not discernible at the wrist. Where the intermittent halt is found with corded arteries, irregular action of the heart, and systemic evidences of cardiac enfeeblement, then it is indicative of structural disease of the heart, rather than mere nervous disturbance of rhythm.

Angina pectoris is commonly found with the advanced condition of cardiac decay, and when so associated, is of the gravest possible character. So important are the relations of the different forms of angina, that the subject must be taken up in a special chapter. It is enough to say here that any sudden alteration of the calibre of the arterioles, by which contraction is caused will lead to a rapid increase of the blood-pressure in the arteries, and so of danger of the left ventricle being arrested in full diastole. This is, probably, the pathology of many cases of sudden death. There is not in fatty degeneration of the heart that labored breathing where all the accessory muscles of respiration are thrown into play, seen in dilatation of the heart with chronic bronchitis or bronchorrhœa, combined with extensive emphysema. The breathing is shallow, with air-hunger (Luft-hunger), rather than oppression. There is, too, an instinctive avoidance of all effort, a peculiar gait and bearing often very noticeable about patients with fatty hearts as they enter the room. They give the impression of being incapable, and consciously so, of any effort.

The symptoms manifested through the nervous system are of great interest. Cerebral anæmia, both chronic and acute, is the common result of impaired arterial fulness. The enfeebled heart can no longer maintain a full pressure within the encephalic arteries perched at the top of the organism, and evidences of brain failure are manifested accordingly.

Consequently, no one will be surprised at the querulousness,

the fussiness, the caprice, the rapid variations of temper, as the blood-pressure in the cerebral arteries alters from time to time—the general evidences of brain impairment manifested by persons with fatty hearts. The vacillating procrastination they exhibit, their inability to decide on matters, their unreasonable and inexplicable conduct, their whims, their preferences and dislikes, are all well known, and are of diagnostic value. They indicate decay of the centre of the circulation as much as brain wasting. In such a condition brain atrophy may be primary, but it is commonly secondary to structural changes in the heart. Then there are acute attacks of cerebral anæmia, not unlike syncope, or, at other times, like apoplexy. Todd, in his "Cyclopædia of Anatomy and Physiology" (article Abnormal Anatomy of Nerve Centres), says: "It (anæmia) is also present when the heart, oppressed by some disease affecting its own structure, fails to propel the blood with its proper force into the brain." B. W. Richardson's graphic picture may be reintroduced here. "The man or woman with a hesitating heart is thereby unfitted for sudden tasks, demands, resolves which, when the heart is firm, are considered of comparatively little moment; for when the heart hesitates, the brain, which reposes for its power on the blood the heart supplies to it, falters with the heart, just as the gas flickers when the steady pressure is taken off the main. From these circumstances some persons who once were known as resolute and determined, lose those qualities when they are subjected to intermittent action of the heart; becoming, as their friends say, uncertain and doubtful in character, becoming, as they themselves feel and know, less the master of themselves, and less secure in their own work, and skill, and power." This striking piece of word-painting should teach us to be more considerate towards elderly persons with fatty hearts, where the arterial blood-pressure is low; to allow for their enfeebled mental processes, and to bear with what is apparent stupidity. This is especially necessary in out-patient work, where persons with fatty hearts seem perversely stupid, failing to grasp the simplest and clearest instructions; and yet, if attentively observed, they are seen to be doing their very best, and are acutely and painfully conscious of their inability to take in what is said.

Of the more acute forms of cerebral anæmia there is a syncopal

form, often seen in very advanced cases, where the patient will sit in his chair and pass through alternate intervals of consciousness and unconsciousness, due, apparently, to some intercurrent disturbance of the heart and circulation. Stokes says, in speaking of fatty degeneration of the heart, "Of the nervous symptoms, the most important are the attacks of apoplexy or pseudo-apoplexy, to which these patients are so liable. This affection differs from ordinary sanguineous apoplexy in three particulars, namely, the frequent repetition of the seizures, the rarity of consequent paralysis, and the fact that there is not only danger from an antiphlogistic treatment, but benefit, both remedial and preventive, from the use of stimulants."

"In some cases the character of these attacks approaches to syncope; and it is difficult to say how much of the affection is produced by the want of arterial, or the stasis of venous blood. The attacks may occur without warning, and the first seizure be fatal. This, however, is rare. In most cases there are numerous seizures at irregular intervals; and in some, sensations referable to the epigastrium and head, having a resemblance to the epileptic aura, give notice to the patient that he is about to be attacked. In some there is a momentary unsteadiness in walking, and in others a tendency to faint, which may be dissipated by any ordinary stimulus; while, in the more decided cases, the patient becomes suddenly comatose, a condition which may be preceded by loss of memory and a lethargic state." After discussing the question whether these attacks are due to arterial anæmia or venous stagnation, he thinks the arterial anæmia the more tenable hypothesis, and writes: "I have noticed one case in which on the occurrence of the premonitory symptoms, the patient, by hanging his head so that it rested on the floor, used to save himself from an attack." This certainly favors the view that these brain symptoms are due to deficiency in the supply of arterial blood.

A curious modification of the respiration is found with fatty disease of the heart; it is known now as "Cheyne-Stokes Respiration;" when first observed it was termed "the phenomenon of Cheyne," but was not connected by him with this special lesion of the heart. "It consists in the occurrence of a series of inspirations increasing to a maximum, and then declining in force and length, until a state of apnœa* is established. In this condition

* Apnœa is not used in such sense now. It is now applied to a condition of

the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspiration.

"The decline in the length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnœa occurs. This is at last broken by the faintest possible inspiration; the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale" (Stokes, p. 324). This is a most interesting phenomenon to observe, and is closely allied in its nature to the fall of the respiration in the rabbit produced by a toxic dose of aconite, and its restoration again under the stimulus of an antagonistic dose of belladonna or strychnia.* The respiratory centre becomes temporarily embarrassed, the respiration falls till the increasing venosity of the blood wakens up the respiratory centre, the blood is again somewhat aerated; then the action of the centre falls until the venosity of the blood is such that respiratory efforts are once more provoked. In such cases it would be very interesting to observe the effects of the subcutaneous injection of atropia or strychnia,—two potent stimulants to the respiratory centre. This phenomenon is not confined to fatty degeneration of the heart. Von Dusch has found it in cases of tumor of the brain, in basilar meningitis, in uræmic coma, and in a grave attack of pericarditis. (Herzkrankeiten, p. 153.) The writer was much struck with this phenomenon when first seen in his student days, and of which he, then, knew nothing. It occurred in a case of sanguineous apoplexy, with rigid arteries, and an hypertrophied heart; the rhythmic rise and fall was well pronounced, and the cycle consisted of about fifteen respiratory acts. The next instance occurred in an old man of eighty-four, with venous congestion, and sopor from primary tricuspid regurgitation.

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super-oxygenation of the blood, so that the respiratory centre pauses for a time, until the blood becomes sufficiently venous to excite it into action.

* See The Antagonism of Therapeutic Agents, the Fothergillian Prize Essay of the Medical Society of London, 1878, awarded to the writer.

There are two certain muscular symptoms of fatty degeneration of the heart, which are worthy of note. The imperfect blood supply to the brain affects the muscular system generally, and gives an uncertainty to the step, and a tottering appearance to the gait; while sudden exacerbations come on, and the patient snatches convulsively at the nearest object of support, lamp-post, stair-rail, companion, or tree. This sudden convulsive snatching is momentary, and passes off as quickly as it came. It is due to sudden cerebral anæmia. The gait of persons with fatty hearts is peculiar; there is a sort of guarded, quiet, distrustful walk, as if apprehensive of the oncome of these attacks; an avoidance of shocks, or effort, combined with evident unsteadiness; there is a want of firmness and confidence in the step and tread, corresponding to what Dr. Richardson has said of the mind. It is readily distinguishable from the various modifications of the gait produced by disease of the brain or cord. There is no irregularity, no loss of co-ordinating power, no jerking of the limbs; neither is there trailing of them, or inability to lift them, or faltering step; there is none of the disarranged toilette, waistcoat wrongly buttoned, trowsers unbuttoned, or shoe down at heel, so characteristic of certain nervous diseases. There is no paralysis of the sphincters, nor trouble with the bladder. From loss of arterial tension, the bulk of urine is small and laden with lithates.

Some changes in the eye, associated with fatty degeneration of the heart, are of value in diagnosis. These are what is termed the "Arcus Senilis," described by E. Canton. It may be complete, or only a portion of the circle, under the upper eyelid, may exist. It manifests itself in two forms, which are quite distinct. In both forms the changes are developed at the point of union of the cornea and the sclerotic tunic of the eye. One form indicates tissue decay; the other is a circular deposit of lime salts, and is usually found with hale old persons. It may be well to take the second form first.

The possessor of the calcareous form of arcus senilis is usually an elderly person, active in mind and body, a person indeed in green old age. Most commonly the eye is of a light-blue color, and the outlook is keen and vivacious. The ring is complete with well-defined edges, and the central portion of the cornea is

clear and bright. The ring may be of a bluish shade, at other times a yellowish-white. This ring is not of bad prognostic omen. It is usually found with hardened arterial walls, and other evidences of calcareous degeneration. But calcareous degeneration, though bad prognostically, is not nearly so bad as fatty degeneration. In the bird a series of bony plates encircle the cornea, and this deposit of lime salts in a circle in the same locality stands in a curious relationship thereto. This is the false arcus.

The true arcus senilis is a ring of yellowish hue, with ill-defined or blurred outlines; a dimmed eye, and often a tremulous gait, are its accompaniments. It may be complete or partial. Its edges melt off into the cornea, so that the line of demarcation is not very distinct. The cornea is cloudy or hazy from fat-globules being scattered throughout its normally transparent tissues; and this cloudiness gives the dimmed look to the eye. When far advanced this eye has a doubtful wavering expression, which is heightened by the muscular unsteadiness. This eye is rarely, if absolutely ever, found without an unsteady pulse, a feeble heart, and other evidences of general decadence. Such an eye at once rivets the attention, and directs it to the patient's general condition, and especially to the state of the heart. The two forms of arcus senilis should never be forgotten or confounded with each other.

The skin, too, is often altered sufficiently for the changes to be noted by the naked eye. At times it resembles discolored parchment, is yellow, dull, opaque, and has none of the glistening which is never absent from the healthy skin. It is not the dry harsh skin of mania, nor the dry, hard, inelastic skin of Bright's disease, seamed with myriads of wrinkles, where the tendency is to calcareous degeneration. At other times the skin is greasy to the feel like new leather, or the outside of a bladder of lard; and the epidermal scales readily rub off, looking fatty and degenerate. When in Leeds, the writer, in conjunction with Dr. Clifford Allbutt, often observed these modifications in cases where degenerative changes were progressing in the viscera, and especially in the heart.

The hair is involved in the change. If fine and silken, it generally falls early from the vertex, and resembles the tonsure of

the monk; a thin fringe extending backwards from temple to temple. Where the hair is naturally strong and coarse it does not fall, but becomes bleached at a comparatively early age; the owner of such a head often being an energetic person of good powers, mentally and bodily. Such is the head of which it is written; "a hoary head is a crown of glory," provided the working of that head is what it should be. The same bleaching goes on in the hair as in the muscular fibres of the heart; in each the pigment matter is gathered together, and in the hair the small masses are aggregated along its axis.

Along with these obvious evidences of degenerative change the temporal artery usually presents peculiarities. It is prominent and tortuous. It may be small and hard, especially where the skin is of a dark hue and the hair only gray, and the organism spare; when so associated it is very sinuous. At other times it is notably thickened, tortuous, and soft to the touch. Such a condition is common with a full habit, a florid complexion, and white hair. Probably here the atheromatous change tends towards fatty degeneration; while in the small, hard, and very tortuous form, the degenerative change is rather calcareous.

Not uncommonly the pulsations of such temporal arteries are visible to the unaided eye. (A tortuous temporal artery is not, however, invariably the accompaniment of degenerative change; it is, at times, seen in comparatively young men, who have worked hard with their heads.) Whatever the condition of the temporal artery, it is the index of the state of the arterial system generally; and the brachial and radial arteries will be found similarly affected. The elongation of the artery on the ventricular systole can often be readily felt; less commonly it is visible. Atheromatous degeneration of the arteries always exaggerates the impulse caused by the ventricular systole, and so often misleads the unwary practitioner as to the state of the pulse. Comparison of the radial pulse with the heart will usually reveal the true state of matters, and show that the hardened artery had created a totally false impression as to the strength of the cardiac contractions. I have known an atheromatous condition of the arteries, accompanied by cardiac hypertrophy, create the impression that the condition called for depressants; had not a frequent intermission, by

its presence, told that there was temporary depression of the circulation. Really, stimulants were indicated; and the ordinary condition was an absence of intermittency.

History and Progress.—The history of fatty degeneration of the heart varies with different individuals. In the majority of instances its initiation is insidious and unmarked, and much progress is made before any very tangible evidence of its presence is manifested; especially in failing hypertrophy. At other times degenerative changes are set up comparatively rapidly, especially in cases where want and privation have struck seriously at tissue-nutrition.

Chronic alcoholism, especially when combined with syphilis, has a most malign influence. The chronic drunkard, who suffers from want of food, is the subject, usually, of a widespread and rapid premature decay.

An abstract of a case from Basham's famous work "On Dropsy," will well illustrate what is meant. The plate which accompanies it is copied from the plate (No. III), which was cut from Dr. Basham's own drawing, and gives the different morbid changes with great clearness. A bricklayer, aged fifty, had been out of work for weeks from failing health, and suffered from want and exposure. He was pallid and œdematous. He had been intemperate. The failure of his health only dated back six months. He was placed on the routine treatment of renal dropsy, and got well and left the hospital. In a year he was back again with dropsy and albuminuria. The heart-sounds were feeble but natural. He was much relieved by treatment, but had constant vertigo and pains in his head. There was also nausea and vomiting. He died of apoplexy, the rupture taking place in the substance of the pons varolii. The arteries at the base of the brain were rigid and opaque, the larger ones being quill-like. The small branch, seen in the plate, was taken from the neighborhood of the effusion. The kidneys were enlarged, pale, and on their cortical surface finely granular. The heart was natural in size, paler than natural in color, the valves healthy. The muscular fibre of the left ventricle exhibited traces of fatty degeneration, as seen in the plate.

"The termination of this case," writes Basham, "illustrates

that fatty degeneration of the kidney is not a partial or local disorder; its presence in these organs implies a greater or less disposition to decay in every part of the organism." It also shows that in widespread tissue degeneration, even a heart becoming fatty may still be powerful enough to rupture arteries still more decayed; and that, too, without any enlargement of the bulk of the heart. The significance of fatty renal tube-casts was greatly insisted upon by Dr. Basham.

DESCRIPTION OF PLATE I.

Chronic Albuminuria—Connection with Degeneration of the Arteries of the Brain and Heart-fibre.

FIG.

1. Cast slightly granular, containing nuclear granules, and two fatty abortive epithelial cells, a few larger epithelial cells natural, and some vesical epithelium.

2.—Slightly granular casts, containing free nuclei, compound granule-cells, and abortive epithelium, some crescentic clusters of nuclei.

3.—Casts becoming less granular, many free nuclei, and compound granule-cells.

4.—The deposit here exhibits the same character as is represented in Figs. 1, 2, and 3. The casts are still more transparent, the free nuclei more numerous.

5.—Heart-fibre from left ventricle—some fibres in a state of fatty degeneration, others presenting the ordinary striæ.

6.—A bloodvessel teased out with needles under water from the neighborhood of the hæmorrhage in the pons varolii. The coats are studded with fat-granules, which remained unchanged in dilute hydrochloric acid but were dissolved in ether.

7.—Epithelial cells from the calyces and pelvis of the kidneys—natural.

8.—Epithelial cells from the fluid squeezed out from the apex of a cone.

a.—Epithelium from a straight tube.

b.—Epithelial cells, fatty and abortive.

c.—Grape-like and crescentic clusters of nuclei from broken-up cells.

9.—A portion of the cortical layer teased out with needles. The convoluted tubes are filled with fine granular deposit, mixed with many free fat-cells. The epithelial cells appear highly granular, and many are detached, or set free from the basement membrane.

10.—Straight tubes from the apex of a cone. The epithelial structure is natural, but the interior of the tube appears blocked up with granular deposit, free nuclei and granule-cells.

11.—Convoluted tubes from the cortex of the kidney. Portions of the basement membrane are denuded of epithelium, and replaced by fine granular and fatty deposit.

Fig. 1.

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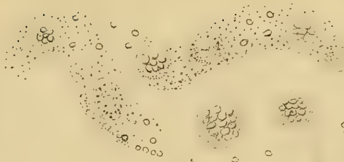

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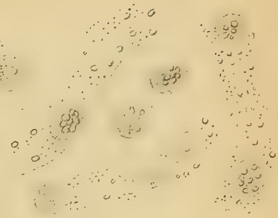

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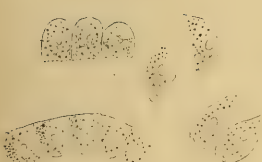

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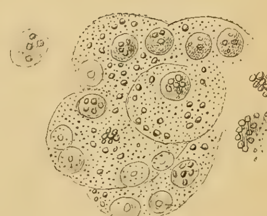

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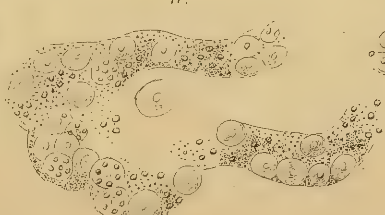

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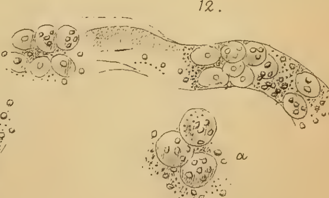

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12.—A tube from the cortical part, showing the manner in which the cells appear to become detached as soon as the canal is set free. The cells appear abortive or atrophic, and are surrounded by a granular material, loaded with free nuclei and fat-granules.

a.—Some large granule-cells impacted in this granular exudation.

At other times a case will present the following features: An anxious uneasy-looking face, with a dim, hazy eye, a discolored skin, a tortuous temporal artery, very prominent, with a bald or gray head; great breathlessness and inability for exertion, a pulse small and irregular with frequent halts; sometimes, indeed, the pulse is scarcely more than a fluttering undulation; there is also difficult locomotion and an impaired and enfeebled intellect. (When such patients present themselves at a hospital they often appear densely stupid, and can scarcely be made to comprehend anything, or retain any order. They seem quite bewildered by the strangeness and novelty of all they see around them.) The heart-sounds are feeble, the first approaching the second in character. Often there is bronchorrhœa. The voice may be petulant or querulous, and there is often a childish impatience or irritability. These patients will be noticed to pant and look distressed when walking, will frequently rest, sitting down, if possible; if not leaning against a lamp-post or clinging to any support.

Or the features of the case may be somewhat different. One of my patients at Victoria Park Hospital was a healthy-looking man of forty-five, a wheelwright. "When first seen, on May 2d, 1876, the first sound of the heart was thin; the second sound accentuated; and there was nocturnal dyspnœa. The diagnosis then made was 'Lithiasis. Failing Hypertrophy.' On July 8th, he was feeling much better; the attacks of dyspnœa had ceased; he felt more equal to exertion; and the first sound was of much better quality, showing increased vigor in the muscular fibres remaining sound and undegenerate." ("Med. Press and Circular," Oct. 25, 1876.) This case was given in a paper on The Successful Treatment of Dilated Heart, to illustrate that something may be done for the worst and most hopeless cases. The treatment was continued, but the aspect of the case grew steadily worse. The chief change was failing power. Locomotion was difficult; if the patient talked as he walked, he had fre-

quently to pause for breath. His respirations grew shallower; his circulation feebler; his general power grew less and less. He could no longer come to the hospital. His daughter came for his medicine. This is her written account of his last days: "He gave up his duties (they were light) about five weeks before he died, and took to his bed over three weeks. But oh, how he labored for breath. His sufferings in that respect were intense, every window and door had to be opened wide; and then we had to fan him besides, and he was obliged to have two to sit with him all night. But when the last came he went off so peacefully. The eyes began to grow dim, yet glassy, and the breath slower and slower until he ceased to breathe on April 27th" (1878). There was no post-mortem, but the case is no doubtful one. There never was any dropsy or any palpitation. Nor was there any dilatation of the heart. These two facts stand in a very suggestive relationship: without change of form the indications are those of failing power. Where there is pronounced dilatation, there are palpitation on effort and dropsy, the indications, indeed, of a dilated heart.

In failing hypertrophy the case may assume the features of a dilated heart; that is, there is irregularity in the volume of the pulse.

Some beats have the character of an hypertrophied heart, being strong; while the bulk are feeble and irregular in volume, and, later on, in time too. Of course, as the case proceeds downwards, the characters of hypertrophy are gradually lost in those of dilatation.

Any intercurrent disease, especially an attack of bronchitis, will often give an impetus to the downward progress and hasten the later stages. The aspect of cases of fatty degeneration of the heart, from failing hypertrophy in their later stages, closely resemble cases of mitral disease; but from the decay often being general, and not local, there are evidences of lung implication, with bronchorrhœa; there is orthopnœa, with, probably, some fatty degeneration of the muscular structure of the diaphragm—a field of much interest, not yet worked at by histologists; the urine is laden with pink lithates, and often contains albumen. To ascertain whether this albuminuria is the result of venous conges-

tion merely, from the failing circulation, or is connected with old-standing renal changes, a careful examination of the tube-casts as practiced by Dr. Basham, is desirable. If the tube-casts are fatty, then the latter is probably the case.

Termination.—Of course the termination of advanced cases is in death either sudden or protracted. Even when the case is taking on the aspect of dropsy and lingering death, a sudden termination is more liable to occur than in cases of mitral disease in young subjects with undegenerate tissues.

Especially is this the case with men. Women are less liable to die suddenly; and in many cases of heart failure in women there is a pertinacious, catlike tenacity to life, till every painful sequel of heart failure is reached, and death takes place at last slowly from carbonic acid poisoning. In one case of sudden death in a woman of forty-eight, death was caused by a dissecting aneurism in the ascending aorta, commencing in an atheromatous ulcer, working down towards the heart, and bursting into the pericardium. The muscular fibres of the heart were the seat of extensive and advanced fatty degeneration. Sudden syncope was found by Dr. Quain ("Lumleian Lectures, 1872,") to occur in twenty-one out of a total of sixty-eight collected cases. That this proportion should be so high can be no matter of surprise when we remember the lessons of the dead-house, and the advanced condition of fatty disease in which some hearts are found. Even when a very advanced condition is not reached, sudden death may be induced by effort, or may occur in sleep. When it comes in sleep, in many cases there may be found a history of effort the day before, or, at times, a heavy supper. Old people are often reticent, and when they have made an indiscreet effort during the day they say nothing about it. But from the known fact that effort, producing cardiac embarrassment (like Clifford Allbutt's experience on the Righi, chapter xiv) during the day, being followed by a like attack at night, we may surmise that sudden death in bed has, in many cases, been preceded by effort the day before.

Then effort at stool is fraught with danger. But the most common cause of death is effort to catch a train or an omnibus. To stand at Cannon Street Station on a Saturday afternoon, and witness the number of elderly men bustling to catch a train at

the last minute, and to note the evidences of tissue degeneration in many of them present, excites surprise, not that sudden death at railway stations is frequent, but that it is not more common, especially as many of those old men, with rotten tissues, carry with them a bag, the weight of which still further embarrasses them and taxes their hearts.

In very advanced cases, changing the patient's linen, or getting them up in order to make the bed, is liable to bring on sudden syncope, either at the time, or some short time afterwards, in bed.

The usual termination of fatty degeneration of the heart is in death.

But it may be questioned if such is always necessarily the case. In failing hypertrophy, with an inelastic aorta, coronary vessels tortuous, and with their lumen diminished by atheromatous change, there can be no prospect of the downward change being turned back; little even of its being arrested for any length of time. The possibility of repair in those cases connected with anæmia, in young subjects, must be entertained. Even in true senile decay such repair may not be entirely out of question. Rindfleisch has described how, in the fatty degeneration of acute fever—myositis typhosa—the muscular fibrillæ are extensively converted into fatty debris, but that when the patient recovers new fibrillæ form from cell-elements already existing within the sarcolemma.

It is from such cell-elements MacKendrick thinks the hyperplasia in cardiac hypertrophy may be developed. In a second plate the growth of these new muscular fibres is shown very clearly. In the bundle most advanced the fatty debris is seen largely absorbed or removed, and the new fibres are quite distinct, occupying a large portion of the space within the sarcolemma; while in the less advanced fibrillar decay, the sarcolemma is seen chiefly filled with debris, and the new fibres but commencing to develop. Such a process then goes on in the heart in the acute fatty degeneration of fever; may not an allied process be possible in some cases of fatty degeneration of a less acute kind? It is not uncommon after a severe illness, or after a prolonged debauch, to find the heart presenting all the features

of fatty degeneration, and yet to recover its tone, its normal sounds and impulse in time. Where this change is due to some

Fig. 37.

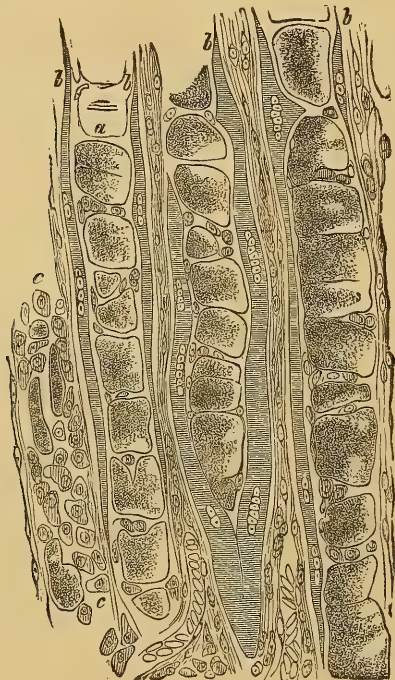
Myositis typhosa. Transverse section $\frac{1}{500}$. *a*, Contractile substance in a state of waxy degeneration. Next it are seen the cells destined for its renewal, which are crescentic in transverse section; *b*, a tube of sarcolemma with a young fibre, circular in transverse section; *c*, another, with a young fibre, which is still semilunar in transverse section, and incloses a gap which might have contained a last remnant of the substance undergoing degeneration; *d*, another, containing a number of typhous cells, together with the remains of the old fibre; *e*, interstitial connective tissue infiltrated with typhous cells.

defect in the fine processes of tissue-nutrition, whose presence is rather detected like a subtle odor, than demonstrable to a skeptical criticism; where there may have been a diet too exclusively hydro-carbonaceous; or tissue repair, the vegetative processes of life, have been temporarily impaired by intercurrent disease; it does not seem impossible that a certain amount of fatty degeneration of the heart fibres may be compatible with repair, by the development of these new fibrillæ, which are shown by Rindfleisch to grow up after myositis typhosa. At all events we are not in a position to say that such repair is absolutely impossible. That excellent physiologist, Dr. Lauder Brunton, thinks that such repair in cases where the fatty degeneration is due to modifications in tissue-nutrition by temporary impairment of protoplasmic power, is very probable, if not certain. Here there are no such arterial changes as positively forbid such repair.

Prognosis.—This is necessarily very gloomy, except in cases of acute fatty degeneration from a sustained febrile temperature. In cases of pernicious anæmia recovery is the rare exception; and

here the prognosis of the heart mischief goes with and depends upon the general state.

Fig. 38.



Myositis typhosa. Fibres seen lengthwise; specimen prepared partly by section, partly by teasing with needles. *a*, The waxy contractile substance of the old muscular fibres; *b*, the young fibres (the shading by transverse lines instead of dots is merely for the convenience of the engraver, and must not be taken to denote the presence of transverse striæ at this early stage); *c*, tube of sarcolemma, containing a very large number of typhous cells, together with slender remnants of the contractile substance. The interstitial connective tissue richly infiltrated with cells $\frac{1}{500}$.

In senile decay prematurely induced, repair is not impossible in all cases, but the large majority, it is to be feared, will go steadily downwards. While of senile decay in very old persons the prognosis is merely a question of time. And it is easy to err about the time; some patients, under apparently unfavorable circumstances, live on surprisingly, especially when no effort, or other accident, intervenes to carry them off suddenly.

In failing hypertrophy the prognosis is not always an easy matter. The early stages of dilatation in a heart, once well hypertrophied, are undistinguishable by the physical signs alone, from a condition of combined hypertrophy and dilatation which has existed for some years, and probably will go on several more years without material alteration for the worse. The whole surroundings of the case must be carefully noted, the condition of the arteries nicely observed, and the other evidences of tissue degeneration accurately appraised before an opinion can be formed as to what form of dilatation we have to deal with, especially where the patient can only make a single visit. The practitioner's acquaintance with the other aspects of heart disease than the mere physical examination, will be tested by these cases, and the insufficiency of physical signs alone to enable him to make a prognosis with any approach to accuracy, will be a matter of which he will become acutely, if not sometimes painfully, conscious. The prognosis will, in many cases, be considerably influenced by the line of treatment pursued.

Treatment.—There are some points to be specially attended to in fatty disease of the heart, which call for mention here. In fatty deposit, or fatty infiltration with general obesity, it may be well to diet the patient carefully with the design of reducing the amount of adipose tissue in the body. In doing so it must be remembered that fat is produced in the body from excess of albuminoids as well as from the hydrocarbons of our food. Then the amount of exercise must be regulated by the patient's powers, so as to avoid the disastrous consequences of overexertion, as seen in the case quoted from Hope, p. 193. The general reduction of adipose tissue is the matter to be arrived at in these forms of the fatty heart.

In fatty degeneration the aim is quite different. Here we wish, if we can achieve it, to remove the fatty debris within the sarcolemma, and foster the growth of new fibrillæ from the cell-elements existing there. Oxidation is necessary for the removal of the effete material; and where there are some prospects of such repair the patient should get as much fresh air as is possible, without fatigue. A certain amount of exercise might be attempted to aid in such oxidation. To increase the oxidizing power of the

blood by the administration of iron, is an idea which suggests itself. But it must be remembered that old people do not tolerate iron very well; and if it be given it is desirable to combine it with alkalies, and administer it in a well-diluted form. Then the food should not be rich in hydrocarbons, and alcohol is peculiarly objectionable.

Nitrogenized food, to supply the material for new tissue elements, must be taken in fair quantity and in easily assimilable forms. Where gout is also present the dietary will manifest considerable difficulties; which will but exist in different proportions in each case. A trace of albumen in the urine will not suggest a dietary rich in albuminoids; nor will a trace of sugar influence the dietary. But albuminuria and glycosuria, no matter how small the quantity of albumen or sugar, are of the most sinister omen when they appear in the later stages of the failing heart. Carbonate of ammonia, spirits of chloroform, tincture of nux vomica and digitalis, with potash or lithia when indicated, are the remedial measures, *par excellence*, in the treatment of the ordinary forms of fatty degeneration of the heart. Where the heart is feeble in elderly persons it is necessary to suspect the possible presence of fatty decay in the walls and treat the case accordingly. The principles, as laid down in the chapter on treatment, must be followed as regards the general management of the case, guided by the details just given.

There are, however, some other matters than those of diet and medicines to be observed in the management of patients with fatty degeneration of the heart, whether in the form of failing hypertrophy or senile decay it matters not, which are of great, indeed paramount importance. The first is the avoidance of effort. If caught in a thunderstorm, away from shelter, the patient must prefer getting wet to the skin to making an effort, even if a change of clothes be not at hand, and he has to go to bed till his clothes get dried. If any accident occurs he must avoid all interference therein; he must strictly mind his own business—and that is to take care of himself; the shock is quite deleterious enough for him. He must avoid bustle and crowds; must be in good time for trains and omnibuses, and must prefer missing them to making efforts to catch them. He must regulate his bowels and not strain

at stool. Sexual intercourse is not without its risks, and may be, and has in well-known instances, proved fatal; though the diminution of the passion reduces this to a comparatively small source of danger, except where the heart changes are found along with locomotor ataxy, when the danger becomes more than a mere hypothetical one. He must avoid all excitement; if a preacher, abandon all great gatherings; emotional disturbance is fraught with danger. Political and other meetings should be shunned for this reason, as well as the air befouled with carbonic acid which limits oxidation. Blood laden with carbonic acid, not only partially anæsthetizes the heart (Cyon), but, according to the late Professor Henderson, of Edinburgh, flows less easily along the bloodvessels. All monetary speculations should be abandoned, the excitement is too dangerous; while the brain, imperfectly supplied with arterial blood, and probably with its own tissues no longer in their integrity, is liable to make erroneous or imperfect calculations, and is no longer suited to complicated mental processes.

The patient's family must be thoroughly apprised that the cerebral working is impaired, and allowances must be made for petulance, perversity, caprice, puerility, or oblique mental processes, which are all outcomes of the physical decay.

All old people are not Nestors; and probably the subjects of tissue decay are never so well satisfied with themselves and their cleverness, as when their mental impairment is quite conspicuous to others. Old people like to think themselves, in mind and body alike, as good as ever they were. It is often necessary to resist their wishes gently but firmly.

Angina pectoris, with its treatment, will be considered in a special chapter. It is, in cases of fatty decay of the muscular fibrillæ of the heart, that breast-pang is so dangerous to life. (See Chapter XI.)

False Hypertrophy.

This is a development of the connective tissue existing betwixt the muscular fibrillæ of the heart. Hayden uses the term "fibroid transformation" of the heart, which he says "is a veritable connective-tissue hypertrophy, or hyperplasia," and due, as Quain

declares, "to chronic interstitial inflammation." It is, indeed, a parenchymatous inflammation, a development of connective tissue, such as is found in cirrhosis of the liver, kidneys, spleen, etc. How it is set up it is impossible to say. Hayden thinks it may proceed from the extension of pericarditis or endocarditis to the muscular structure of the heart.

At one time it was held that hypertrophy of the heart was due to the development of unstriped, fusiform fibres, of ordinary involuntary muscular fibres indeed; but now we know it is due to the development of new fibres from the protoplasmic masses existing within the sarcolemma. By reference to Fig. 38 it will be seen that the growing fibre is fusiform in shape. True hypertrophy is a hyperplasia of the muscular fibrillæ; false hypertrophy is a hyperplasia of the connective tissue betwixt the fibrillæ. True hypertrophy adds to the heart's power; false hypertrophy adds no strength, rather it diminishes the heart's power. Sir William Jenner has described this form of heart change. The walls do not collapse when cut, but are resilient and recover their shape when bent, like a piece of an india-rubber ball. Of course the contractile capacity of muscular walls so altered can be but little.

There are no signs or symptoms peculiar to such change. There are the obvious evidences of increase in bulk; and the absence of evidence of increase in power, in the shape of a firm, incompressible pulse. Dr. Broadbent thinks that in this form of disease the heart-sounds approach a "tick-tack," rather than the normal sounds. But the diagnosis must always be a matter of suspicion rather than certainty.

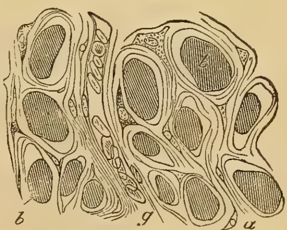
There are no reasons for supposing that any antiphlogistic regimen or remedies could influence such connective-tissue growth. Nor, on the other hand, can we suppose this development of connective tissue to be due to venous congestion, as is the case with the enlargement of the liver, spleen, and kidneys in cases of heart failure, where there is fulness of the venous radicles. The arrangements of the coronary circulation are opposed to such hypothesis. The heart-walls squeeze the blood out of themselves in their contractions, and the oblique manner in which the coronary veins perforate the walls of the right auricle prevents regurgitation into

them. If false hypertrophy of the heart were due to venous congestion it would be present in most cases of heart failure, instead of being of comparatively rare occurrence. Hope says, "This affection is very rare."

This connective-tissue hypertrophy may greatly increase the bulk of the heart, as seen in the large heart in the museum of St. George's Hospital, which weighs forty ounces; the increase being due to the growth of connective tissue, not of muscular fibre.

This growth may not be general. Rokitansky writes: "*Induration* is a mode in which inflammation frequently terminates; it is often found to have occurred at some isolated spots, especially in the heart. The exudation in the inflamed part coagulates and becomes converted into a whitish, lardaceous, firm callus, which assumes a fibroid structure, but is still traversed by a few pale and broken muscular fibres. The appearance of the callus varies according to the original quantity of exudations; at first it forms cords and streaks which ramify amongst the fibres and fasciculi of the muscle, or more extensive round, or nearly round masses, which may be tolerably circumscribed, or may branch out irregularly in various directions. In the course of time it may diminish considerably, partly from absorption, partly from its shrinking and becoming more dense, and from the disappearance of the muscular substance that still remains within it. As it thus diminishes in size, it draws in the surrounding tissue, and assumes the appearance of a deep cicatrix. Such cicatrices have received the name of the scars of Dittrich."

FIG. 39.



False Hypertrophy.

a. Excessive Connective Tissue.
b. Compressed Muscular Fibre.
g. Capillary full of Blood.

The manner in which this connective-tissue hypertrophy affects the muscular fibrillæ is well shown in this plate from Rindfleisch.

He calls this development of connective tissue compensatory to the wasting of the primitive bundles of muscular fibre. It is more probable that the wasting of the muscular fibre is due to the compression exercised by the growth of the connective tissue.

This connective-tissue change is rather a disease of the dead-house than a clinical matter. Probably it is seldom distinguishable in life from fatty accumulation or fatty infiltration. Dr. John Fothergill published a case of this fibroid change in 1774. It occurred in a patient sixty-three years of age, short, and of full habit, who had had fits of angina for three years, and who, ultimately, died suddenly in a fit of anger. The post-mortem was made by John Hunter, who stated, "The heart, to external appearance, was sound; but, upon examination, I found that its substance was paler than common, more of a ligamentous consistence, and in many parts of the left ventricle it was become almost white and hard, having just the appearance of a beginning ossification." (Hayden.)

Atrophy.

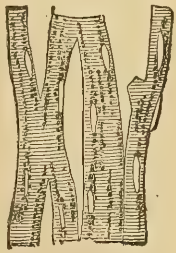
Atrophy of the heart, that is, a diminution of its bulk, is found in all wasting diseases, as phthisis and marasmus, however caused. Walshe says, "It appears sometimes to follow the tight embrace of pericardial false membrane; to depend occasionally on narrowing and calcification of the coronary arteries." Hayden says that, "The pulse in all cases of atrophy of the heart is small, quick, all but inappreciable; but in those forms in which it is unassociated with fatty or other tissue-change, in an advanced stage, it is regular. The area of pericardial dulness, both deep and superficial, is contracted, and the cardiac impulse is limited and feeble. The sounds are sharp and clear when the structure of the heart is sound; but where it is softened the first sound is dull, faint, and scarcely audible."

Virchow has found the heart and aorta small in certain girls who seem liable to become the subjects of anæmia; and L. Beale, in his work on the urinary organs, speaks of a class of persons with small hearts, congenitally, small bones, and general plumpness, who seem to give way to kidney disease and a general break-up at an early age in a strange manner. The prognosis will depend upon the associations; and the treatment is that of the associated state.

Brown atrophy is an affection first described by Rokitansky. It consists of a development of pigment within the primitive

muscular bundles, and extends throughout the heart. Whence this pigment is derived is not known. Brown atrophy is found with advanced life, and with cachexiæ, not necessarily connected with old age. The accompanying plate is from Rindfleisch.

Fig. 40.



Brown Atrophy.—(After Rindfleisch.)

Larcher and Ducrest have drawn attention to the enlargement of the heart in pregnancy. After parturition the uterus undergoes involution by fatty degeneration and absorption of the muscular fibrillæ, until the unimpregnated bulk is again reached. Sir James Simpson taught that sometimes the process goes on until the uterus is lessened to below its normal size. Dr. A. Wiltshire holds that in some women the reduction of the bulk of the heart after parturition goes on till a certain amount of atrophy is reached.

The subject is one of great interest, but is still very obscure.

Amyloid Degeneration.—This is a rare form of disease of the heart-walls. According to Oppolzer, it consists of the formation of a shining, colloid-looking material within the primitive bundles. It gives the usual reaction of amyloid matter to iodine and sulphuric acid, which is not a true blue. It is usually found in the right ventricle, especially when the right ventricle is enlarged (Rokitansky). It has no special symptoms, but is usually found along with enlarged amyloid liver, spleen, or kidneys, with albuminuria and tube-casts. Though I have taken much trouble, in Vienna in 1871–2 and since, I have never succeeded in procuring a microscopic section of this form of tissue-change.

Syphilitic Gummata.—These are found in the hearts of the subjects of constitutional syphilis. It has been termed syphilitic myocarditis, but these growths do not spring from the muscular fibrillæ, but from the interstitial connective tissue. According to Virchow, these growths consist of fine granulation-corpuscles, which not uncommonly undergo fatty degeneration into cheesy masses, at one time mistaken for tubercles. At other times these gummata become converted into fibrous masses of connective tissue.

These masses are scattered throughout the substance of the heart, just as gummata are in other muscles; and are found from

the size of a bean to a pigeon's egg (Virchow), or a billiard-ball (S. Wilks). They are often accompanied by a thickened condition of the endocardium and pericardium; and may be seen showing through the columnæ carneæ, under the thickened endocardium. They have been described by Lancereaux and Ricord, and formed the subject of a notable discussion between Virchow and Von Barensprung, when Virchow demonstrated they are not tubercular masses. The largest are found in the septum ventriculorum (Von Dusch), and project from thence into each ventricular chamber. These growths, in the interventricular septum, have been supposed to be a cause of perforation of this septum in children congenitally syphilitic by some; but this has not been proven.

As to the treatment of these growths it is that of syphilis, and Lancereaux found the symptoms relieved by iodide of potassium. But the treatment must be resorted to early to be of service; it is futile when once the growths have undergone caseous degeneration, or been converted into fibrous masses. How the diagnosis of such gummata is made is not known to the writer.

Tubercle.—The true miliary, or gray tuberculosis of Laennec, is found in the connective tissue of the heart-walls in cases of acute tuberculosis.

Cancer.—Cancer has been found to attack the heart, in the forms of carcinoma, medullary, or melanotic cancer (Virchow). It is commonly secondary, but may be primary. Bodenheimer has described a case of primary nodular cancer of the right side of the heart.

Parasites.—The heart is liable to be the seat of parasites, or entozoa, the cysticerci, both cellulosa and echinococcus. In Amoy dogs commonly have balls of a form of filaria in their heart chambers.

Polypi.—There are true polypi and false, which last are fibrinous clots usually formed in the death agony. Grisolle has collected authentic cases, and Flint ("Diseases of the Heart," p. 287) describes a case which occurred in Bellevue Hospital. In this case there were two polypi, both growing from the right auricle, one extending into the right ventricle. W. T. Gairdner has described a case of polypus of the right auricle, which caused a presystolic murmur, and the case was supposed to be one of tricuspid obstruction.

CHAPTER IX.

SOME CONDITIONS WHICH SIMULATE ORGANIC DISEASE OF THE HEART.*

THE gravity which surrounds all cases of organic disease of the heart, and especially of its walls, gives certain simulative conditions great importance in the eyes of careful practitioners. They know that if they fail to recognize the presence of organic changes, not only do they expose themselves to the risk of their professional knowledge being called in question on the discovery being made, but that the patient and his interests may and probably will suffer in consequence. If such disease exist and be not detected, the patient may pursue a course of life most prejudicial to his chances of existence and to his material interests. In my own experience, I have found it often a matter of the greatest moment and withal of difficulty, too, to answer the question put by the patient: "What are my chances of life; as I shall arrange my business affairs accordingly?" It is clearly the duty of the medical adviser in such cases to be very careful, as on his answer may hang the fortunes of the patient's family. Even where pecuniary interests are not implicated, the patient has a distinct claim on our utmost consideration, as his chances of life may rest largely on the answer given. By care life may be prolonged; by the absence of proper care life may be cut short, even when it is clear that under the most favorable circumstances it cannot be very prolonged.

On the other hand, much misery and much injury may be done by an erroneous diagnosis in the opposite direction. If the medical adviser thinks that there are grounds for suspecting fatty degeneration, he may give advice which may be very pernicious. He may disturb the patient's mind by creating the im-

* This short chapter was read before the West Kent Medical Society, January, 1878. It stands in its primitive form.

pression that he is the subject of an insidious disease of the gravest character, liable to terminate very suddenly, and so make him wretched without reason. He may cause the patient to arrange his monetary affairs in a way that shall be very detrimental to his interests. In fact, for every thinking and conscientious practitioner, the question of the presence or absence of structural change in the heart-walls is one of the utmost gravity.

Da Costa writes: "There is as yet no sign discovered by which we can say that the dangerous disorganization (fatty degeneration) of the muscular fibres of the heart is in progress. We may, however, suspect it, if the signs of weak action of the heart—feeble impulse and ill-defined sounds—coexist with a pulse permanently slow, or permanently frequent and irregular, and be met with in a person who is the subject of a wasting disease, or who has arrived at a time of life at which all the organs are prone to undergo decay. Something more than a suspicion is warranted, if, in addition, there be proof of fatty degeneration elsewhere, such as an *arcus senilis;* or if it be ascertained that the patient suffers from paroxysms of severe pain in the heart, that he sighs frequently, that he is subject to seizures during which his respiration comes to a standstill, and that he is liable to be stricken down with repeated attacks having the character of apoplexy, save that they are not followed by paralysis." Such then are the indications of fatty degeneration of the heart; if also there be tortuous and atheromatous arteries, a skin dry and wrinkled, or smooth and unctuous, little doubt can remain.

In this age of physical examination, when a diagnosis rests mainly and often exclusively upon physical signs, the subjective phenomena are regarded as of secondary importance; if, indeed, they be not rarely put out of court as witnesses whose testimony is of no importance. With the physical signs as his main guide, the practitioner, when brought face to face with certain conditions, is often in a very difficult position when called upon to answer the question, "Are the heart-walls sound?" To this momentous question it is confessedly difficult, in many cases, to make any answer with confidence; and yet it is of stupendous importance to the patient to have an answer, and still more a correct one.

Such being the case, it may be well to review those conditions

which simulate fatty degeneration of the heart, and which are liable to be taken or mistaken for it. In the first place, there is a condition of temporary debility of the heart, which is the most common source of error. Stokes goes so far as to say, "It would be difficult or impossible to draw a line of distinction between the signs of simply weakened heart, and this condition combined with fatty degeneration." And this difficulty is experienced by all; and if so great a diagnostician as Dr. Stokes has felt the difficulty, it is no wonder if an ordinary mind occasionally falls into error. Certain it is that there are times when one feels that, so far as the physical signs go, it is impossible to get rid of the fear that there is fatty degeneration in the case. The lack of impulse, and the faint character of the first sound, are very suggestive; and when to these are added breathlessness upon exertion, inability to sleep with the head low, and a readily compressible pulse, the case looks dark indeed. There is certainly present cardiac debility; there may be also present structural decay. How is the presence or absence of the latter to be made out? Certainly not by the physical signs. We are left then to the surroundings of the case, and these will not always release us from our difficulties. When these signs are found in young anæmic women there is no great difficulty. Walshe says, "This state of things, which I have principally seen in young females, and often in connection with disordered menstruation, is curable by attention to the states of the uterus, and by the tonic invigorating plan of treatment." It is clear that in conditions of general malnutrition of the muscular system the heart must be involved; and its imperfect nutrition will manifest itself by evidences of debility and functional impairment. In such cases, the well-known absence of structural disease, as demonstrated by experience in the dead-house, will suffice to settle the question without much doubt.

We know, too, also from pathological experience, that the heart is much weakened in fevers, especially relapsing and puerperal fevers. Indeed, a condition of acute fatty degeneration is produced by a very high temperature maintained for any length of time. Patients recovering from relapsing fever sometimes die suddenly when walking about the wards of fever hospitals from sudden failure of their weakened hearts. But in these cases

there is nothing special, nothing different from the other cases which recover; unless it be in the amount of softening which has gone on in the heart. The heart structures, then, are liable to become degenerate in acute disease, and from this condition they recover as the general convalescence progresses. This form of temporary debility of the heart, with its accompanying signs and symptoms, also is free from any great difficulty in diagnosis. But there are forms of cardiac asthenia where it is next to impossible to say whether there is irremediable degeneration present or not. They occur in elderly persons whose arteries are far from being above suspicion; where the *tout ensemble* would almost favor the view of fatty degeneration being present. In my experience the case is most frequently of this kind. An elderly person is not very well and calls in medical aid. The complaint is of general languor, and malaise, loss of appetite, disturbed sleep, some thirst, dyspnœa experienced on any exertion, and a certain shortness of breath. There is no pyrexia, and no obvious disease. The pulse is found to be feeble and readily compressed, and it is affected by assuming the erect posture. Naturally, the heart is examined. Its impulse is felt to be absent entirely, or if present, very feeble. The first sound is very slight and distant, and less audible than the aortic second sound; it also can only be heard over a very limited area. These signs, taken along with the subjective symptoms, will make any man hesitate and doubt. It is prudent, certainly, to be cautious in such a case and give a guarded answer. Only time and the effects of treatment can demonstrate that the suspicion of fatty degeneration is not well founded, and that the case is being misinterpreted. One very striking case in point occurred to me some time ago in an old gentleman whom I knew well. For my life could I be sure whether his case was one of cardiac asthenia only, or that fatty degeneration was present. However, the general condition was treated by rest in bed, suitable food, and the administration of tonics. In time the heart's sounds became normal, and not only that, but a distinct aortic systolic murmur became audible, and remained so long as I attended him, and was found by the medical gentleman who attended him when I left the neighborhood. When the old gentleman recovered his

usual health, it was found that there was a fairly good hypertrophied heart, which for several years longer struggled so successfully against the aortic stenosis as to permit him to walk about his garden and enjoy life.

At other times, conditions of cardiac asthenia, presenting the physical signs of fatty degeneration, may arise from sedentary labor of a prolonged character, combined with too short hours of sleep. Some four years ago a well-marked instance of this came under my notice. A gentleman, engaged in a business which did not call for much physical exertion, but which entailed long hours of desk-work carried far into the night, found himself growing scant of breath, and only able to walk slowly, especially after a meal. He was a stalwart man, weighing some fifteen stones, gray-haired, and gouty. His heart-sounds were very faint and feeble, and the impulse could not be felt through his massive chest-walls. His pulse was fairly good, being exaggerated by atheromatous vessels. A suspicion of fatty degeneration was not ill-founded in such a case, and, indeed, a diagnosis to that effect had been made by an eminent physician whom he had consulted. Yet when I saw him I felt great doubts as to whether any fatty decay was really present, in addition to a condition of asthenia. On going carefully into his habits, and finding that he only had a few hours' sleep each night, he was advised to cut down his personal work, and have longer hours of sleep with more exercise. In a short time he lost his disagreeable symptoms, and is now as well as a man of his years, about fifty-five, can well be. It might be maintained that in this case some fatty change was actually present and removed by the means described at p. 227.

Another form of senile change is also the source of diagnostic error, viz., that of ossified costal cartilages, with emphysematous lung behind them. Here, too, the heart's impulse is imperceptible, and the sounds are distant and feeble; while to percussion little or no heart dulness can be made out. A condition of fatty degeneration is thus simulated, but not very closely. Nevertheless, it has led to mistakes, and a striking illustration occurred to my friend Dr. Clifford Allbutt, of Leeds. One day the wife of a gentleman in a neighboring town came to him hurriedly, and

desired him to go with her immediately to see her husband. He had suffered from asthmatic dyspnœa for some years, and in the morning, as he did not feel well, and was intending to go up to London that day, he thought he had better call in his family medical attendant. This gentleman made a physical examination of his chest, and pronounced him to have a fatty heart. Another opinion was also called in, and the original diagnosis corroborated. The journey was forbidden, and strict quietude advised. The patient's wife was very much perturbed at this, and hoping that the diagnosis might be erroneous, set off for Dr. Allbutt. When he saw the patient he admitted the physical signs, but pointed out that the pulse was firm and incompressible, and that, in all probability, there was a large and structurally sound heart behind the emphysematous lung and the rigid costal cartilages. The local medical men handsomely apologized to the patient for their mistake, and the journey to London was proceeded with without mishap. Such a mistake could only take place with practitioners who had been taught, or had taught themselves, to rely exclusively on physical signs, which, however useful ordinarily, on this occasion betrayed them into error.

A much more common condition than any yet described, and often of a still more puzzling nature, is that presented by women at the change of life. Here the condition closely simulates fatty degeneration, and in many cases, in all probability, there is a blended condition in reality. But much more frequently the condition is one of passing asthenia uncomplicated by structural decay. Such patients are most common in the upper classes. A typical case would present the following features. A lady of indolent habits, not averse to the pleasures of the table, with a taste for tea, and inclined towards less innocent stimulants, but not to any reprehensible length; stout and not rarely pallid, with white and slightly œdematous-looking hands,—complains of shortness of breath on any attempt at exertion. She also complains of waking in the night with dyspnœa, and of inability to sleep with her head low, and the necessity for an extra pillow or two. On examination, the pulse is found feeble and easily compressible; the heart's impulse cannot be felt, and its sounds are thin, weak, and distant, while its rhythm is disturbed somewhat.

It is impossible by percussion to determine its exact size, but there is every reason to believe that it is somewhat dilated; and it is undoubtedly feeble. The patient complains that she is liable to palpitation on slight effort, or any mental excitement. Palpitation is common, too, at night, and is always present when she is awakened by dyspnœa. But there is another phenomenon which is felt to be much more distressing, and that is a feeling as if the heart was stopping, or, indeed, had stopped, which is most alarming. Such patients declare that when they feel their hearts palpitating, they are comparatively comfortable, for they know their heart is beating; but that the feeling of stoppage causes acute alarm. In such cases the appetite is defective, and the digestive power low. The bowels are confined, and there is a certain amount of flatulence present. Accumulation of gas in the stomach or in the transverse colon, pressing on the diaphragm and so displacing the heart, causes that organ to beat with difficulty, and so evokes palpitation and dyspnœa. These patients, too, are liable to syncopal attacks, such as are found in actual fatty degeneration. They readily perspire; they have cold extremities, and, indeed, present the whole array of symptoms which Dr. Hayden gives as those of fatty degeneration. In these cases, from inability to take exercise, from their occupying warmly-heated rooms in consequence of their defective production of body heat, there is undoubtedly defective oxidation of the tissues. From their impaired digestion and loss of appetite there is defective nutrition. The heart, an organ ever in action, is certainly badly nourished—worse nourished, perhaps, than any other part. From the imperfect oxidation, it is very probable that there may be an accumulation of fatty debris in its structure as well. It is impossible to be absolutely certain that such is not the fact in any case before one; in many it is more than probable, in some it is certainly the case. Where there is fatty degeneration blended with the weakened condition of imperfect nutrition the case proceeds steadily downwards, the symptoms deepen in gravity, and the end is death.

But in the large majority of cases a less disastrous termination is the result fortunately. The ill-health of the menopause passes away in time; the nutrition improves, and with it the state of the

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heart-walls; and, after a time, the invalid is found to enjoy fairly good health, and to be capable of a considerable amount of exertion, and little is heard of the once troublesome heart—indeed, the patient is no longer an invalid. Having passed through a period of ill-health and some danger, the organism passes into the placid condition of post-catamenial existence; little disturbed by the rhythmic periods which constitute the refrain, as it were, of the catamenial cycles. The appetite returns, and the heart is well nourished. It is free, too, from the reflex disturbances to which it was subject during the menopause, and which often occasioned the fits of palpitation and the syncopal attacks. The recurrent attacks of pallor or of flushing have gradually ceased; and, indeed, the vascular system is once more free from the perturbations to which it was for a time subject, and which were so alarming and suggestive of serious organic disease.

There is no doubt about the fact of the reduction of a simply dilated heart to its normal size, or near it. And where there has been a condition of debility with dilatation merely, and the heart-walls have been structurally sound, such patients recover perfectly. But when there is also some molecular decay the case progresses less satisfactorily. It is probable that a certain amount of fatty degeneration is not incompatible with repair, as seen in the softened hearts of relapsing fever. How far such decay extends is a matter for the most attentive watchfulness and careful calculation in each individual case. As to what is the exact condition in every case for diagnostic and prognostic purposes, can only be ascertained by painstaking examination and by accurate estimation of each factor in the case. The case of the late Harriet Martineau illustrates what has just been said. Though Sir Thomas Watson and others diagnosed the case correctly, it is clear that a mistake was made by some others, and that the lady herself was under the firm impression that she was the subject of organic disease of the heart. Yet she lived more than twenty years to refute such a diagnosis. The fact that the first sound was described as "noisy," should in itself have put those who examined her on their guard as to the improbability of fatty degeneration in the case. She was at a certain time of life. She had an enlarged ovary as a source of irritation, and her heart was disturbed reflexly.

When the ovary escaped out of the pelvis into the abdomen the perturbations, reflexly excited, ceased, and the mesmerism got the credit of it. That Harriet Martineau died ultimately of heart disease is very probable. But the reflex disturbances when she was fifty-three years of age, surely bore no relation to the disease which existed when she was seventy-four.

As to the murmurs, hæmic or dynamic, which simulate the murmurs of valvular disease, little need be said. The hæmic murmur of anæmia is well known; it is situated in the pulmonary orifice, and that is in itself very nearly sufficient to establish its non-organic nature. So also the pulmonary murmur heard when the lung does not sufficiently cover the heart, and aggravated by expiration and lessened by inspiration, speaks for itself. The dynamic murmurs heard at the mitral orifice, which are systolic in time, and which have been so carefully described by Dr. George Balfour, are certainly difficult to distinguish from those of mitral regurgitation, and cannot so be distinguished by the signs revealed to auscultation or percussion. The general features of the case are the only guides to a correct diagnosis; and it is just the general features of each case, rather than the physical signs, which must determine whether there is present actual organic disease of the heart, or merely a condition which closely simulates it.

CHAPTER X.

RUPTURE OF THE HEART—TRAUMATIC INJURIES—DISPLACEMENTS—IN OVARIAN DISEASE—STRAIN AND SHOCK.

Rupture of the heart from disease of its walls is a not very common accident. It may occur from embolism of a coronary vessel, from fatty degeneration, from an abscess, or from an ulcer formed by the breaking down of a syphilitic gumma, or from an hydatid cyst. It is most commonly associated with old-standing disease. The walls of the heart may be the seat of extensive disease, which may be more marked at some points than others. Consequently when the heart-walls contract the internal pressure on the diseased spot is more than its unsound structure can bear, and a tear in the texture is the consequence. Schroetter thinks that the time of rupture is "the moment of systole, and at its commencement, when the pressure within the heart is at its greatest." The locality is most commonly the left ventricle, and much more frequently on the anterior than the posterior portion of it. Then, but at a long distance, the right ventricle, then the right auricle, and last the left auricle. Thus of Elléaume's fifty-five cases, forty-three occurred in the left ventricle, seven in the right ventricle, three in the right auricle, and two in the left auricle (Schroetter).

The pathological associations of rupture are fatty degeneration with disease of branches of the coronary arteries. Some twigs are more diseased than others, and thus lead to spots of advanced softening; or an embolism, as a piece of atheromatous tubercle at the orifice of a coronary vessel, may block up a small artery and lead to acute fatty degeneration of a localized patch of the heart-wall. The other causes are comparatively rare. As to the rupture itself it is usually a fissure following the course of the muscular fibres, irregular in its edges, and often containing coagula. The fissure is usually small, but may be extensive.

There may be one or more, and they may not occur altogether; one fissure may contain a clot, and even an attempt at repair may be instituted.

The rupture may be complete at once, or it may progress through layer after layer, like a dissecting aneurism. These ruptures are regarded as usually extending from within outwards. Blaud thought, from the external opening being greater in some cases than the internal opening, the tear might take place from without inwards; and my colleague, Dr. Alfred Wiltshire, met with a case where the rupture was from without inwards, and imperfect slits were found in the outer surface of the heart. From this it would appear that the cause of rupture is not always the internal pressure on a decayed spot, but that the external fibres may be torn in contracting. It is not probable that rupture is caused by distension of the heart-walls in diastole; but rather by centripetal pressure during systole, or, more rarely, by contraction of the external layer of fibres.

As to the time of life at which it occurs, of Quain's collected cases, two-thirds were over sixty years of age. From its connection with fatty degeneration it is probably more common in the male sex.

As to its production, rupture has occurred during sleep, when it is probable that the fissure commenced in some waking effort, and merely perforated the cardiac layer of the pericardium during the quietude of sleep. It is usually associated with effort, as running to catch a train, an active effort, as lifting a weight, an epileptic attack, effort at stool (Barth, of twenty-four cases, found five so associated), the act of coition, or even the arrest of the peripheral circulation in a cold bath (Von Dusch).

The symptoms are sudden pain and collapse. Where death is not instant, there is nausea, fainting, and vomiting. The skin is cold, the pulse is small and irregular, and almost imperceptible. The appearance is that of syncope. As the pericardium becomes distended with blood there is increased cardiac dulness. The heart-sounds are diminished.

Though certain attempts at repair have been found, the termination is practically always in death. But the patient may live several days—in a case of Barth's eleven days elapsed before

death took place. As to the treatment, that must entirely depend upon the circumstances of each case. Stimulants are indicated; but it may be questioned how far they may not increase the rupture. Narcotics, to relieve pain, may be demanded.

Traumatic Rupture.—The heart may be ruptured by external violence, like other internal organs. Falls, violent shock, or compression, as by the buffers of a railway carriage, may occasion it. In traumatic rupture the thinner portions of the heart are usually the seat of rupture; being the reverse of rupture from disease. The heart has been torn from the aorta without external injury to the body.

Wounds of the Heart.—These are, generally, immediately fatal, but not necessarily so; nor, indeed, are they always fatal. In extensive injury death takes place from shock, the heart's action being instantaneously arrested; at other times it takes place from hæmorrhage. A case is recorded where a rapier passed through the left ventricle and septum into the right ventricle; yet the patient lived seventeen days, losing about a pound of blood daily. At other times the wound may heal. D. G. Fischer, of Hanover, has collected 452 cases of injury to the heart; of these no less than 123 involved the right ventricle, which, from its position, is most liable to injury from a wound in the anterior aspect of the thorax. Of these wounds no less than 50 healed; and of these 50 cases no less than 33 were the subjects of autopsy, establishing the fact of healing beyond all question. "Injury of the auricle is always fatal. So, also, there is no recorded case of the healing of a rupture, but, on the other hand, according to Velpeau, even a wound which penetrates the entire thickness of the ventricular wall may heal by cicatrization. The direction of the wound will be of the very greatest importance as regards a cure—the more oblique the canal, the more favorable will it be; but even very fine and very oblique wounds have proved rapidly fatal. In these latter cases possibly the injury of one of the great vessels hastened the fatal termination. Yet in a patient thirty years of age, it was shown that it is possible that a coronary artery, which has been cut across, may heal. Larrey" (Schroetter).

Prognosis and Treatment.—Of course the prognosis of all cases of wound of the heart is very bad. Shock is grave and may kill.

At other times the shock is slight, and the patient rallies to encounter the risks of hæmorrhage. Syncope tends to arrest the bleeding, and should not be rashly interfered with. Probing the wound is necessarily dangerous, and is not desirable. Schroetter writes: "Foreign bodies, such as needles, the point of a knife, etc., remaining in the wound do not, strange as it may seem, render the result more unfavorable; indeed, we have quite a number of authentic cases where recovery took place with the foreign body remaining *in situ*." The treatment is that of perfect quietude; if the pulse is at all hard, to bleed the patient freely, and to give narcotics freely where there is much pain or restlessness. The removal of the foreign body is not always indicated.

Foreign bodies have found their way into the heart by the œsophagus. "Entering in this way they may be completely imbedded in the wall of the heart, partially project into its cavity, or lie free in the same, and become firmly fastened *in situ* by a sheath of fibrin or connective tissue, or, finally, may pass entirely through the heart" (Schroetter).

The general impression that wounds of the heart are necessarily at once fatal is erroneous. Doubtless they usually are; but the above account demonstrates that injury of the heart may be recovered from. Foreign bodies have been found in the heart which have been there long before death, and which had nothing to do with the death of the individuals in whom they were found.

As to the treatment of wounds of the heart, it is obvious that all surgical interference is out of the question. The patient must be kept perfectly quiet; in syncope lies a prospect of a clot forming in the wound, especially if an oblique one, and no attempt to rouse the patient out of the syncopal state should be essayed. Then, afterwards, it might be necessary to give aconite or chloral hydrate to calm the vascular system, and the system generally.

Displacements.—The heart is not absolutely fixed in its position by its attachments, and is displaced under certain conditions, as by a fall or other violence, by disease, or it may be congenitally on the right side and not on the left. A case is related by Cheyne, of Dublin, of displacement of the heart to the right side, occa-

sioned by an accident. It remained there, palpitating a good deal, till it got accustomed to its new quarters. Here, probably, simple rupture of the pericardium must have allowed the heart to escape, while the elasticity of the vessels was insufficient to draw it back to its primitive place ("Cyclopædia of Practical Medicine").

Congenital displacement of the heart is a very rare condition. It may be found without the usual displacement of the viscera, as the liver being on the left side and the spleen on the right. Stokes (p. 463) relates a case of a heart which was on the right side, and where a correct diagnosis was made. Some few years ago, Dr. A. E. Sansom showed, at the Medical Society of London, a patient with mitral regurgitation, where the heart beat on the right side congenitally, and the murmur was heard extending to the right of the right nipple. Indeed, it had just the usual characteristics only on the right and not on the left side. Cases of transposition of the viscera are to be found in all large pathological museums. Two such cases are to be seen in the museum of the Pathological Institute of Vienna.

Displacement by disease is comparatively common. Left pleuritic effusion usually thrusts the heart over to the right side of the sternum. Right pleuritic effusion, which is rarer, sends the heart further to the left than it is normally. If pleuritic adhesions form it may be permanently imprisoned in its new locality. It may also be displaced by an aneurism, by an emphysematous lung, by cancer, or by diaphragmatic hernia (Da Costa.)

It is also displaced by several abdominal conditions, as ascites or enlargement of the liver. The most important of all such forms of displacement is where the cause is ovarian disease. It is desirable to ascertain the condition of the heart in reference to the performance of the operation as well as the administration of anæsthetics. Recognizing this, I have asked Mr. Knowsley Thornton to describe the condition for me, and he has kindly done so. He writes:

"Large cystic tumors of the ovary or collections of fluid in the peritoneal cavity, surrounding ovarian or other abdominal tumors, may cause displacement of the heart, either directly or indirectly.

"The direct displacement is, primarily at any rate, a vertical one,

the heart being pressed upwards along with the other thoracic organs by the diaphragm. In this form of displacement the ribs are pressed outwards, and the normal curve of the diaphragm is destroyed. In the first instance, it is obvious that the lungs are most affected, but eventually the whole contents of the thorax are pressed upwards, and as the upper boundaries of this cavity are rigid, the lungs are compressed; while the less yielding heart is pressed upwards as a whole, and, as a consequence of its position and attachments, its apex is pushed outwards towards the left side. In other words, a certain amount of transverse displacement occurs along with the vertical one.

"This direct displacement must be excessive before it gives rise to serious inconvenience; and it nearly always becomes associated with some form of indirect displacement, to be referred to farther on, before it gives rise to well-marked thoracic symptoms.

"The symptoms arising from direct displacement vary, of course, with its extent; and they are of a general rather than special kind. The following will usually be present in a more or less marked degree. Shortness of breath on extra exertion, quick movement, climbing upstairs, or walking up a hill, and on attempting to lie down flat in bed. Such cases are always more comfortable when moving quietly about; more so even than when sitting still or partially reclining, for the simple reason that the tumor, or free fluid falls or rather presses downwards and forwards, stretching the elastic abdominal parietes, and causing a more or less pendulous condition of the lower part of the abdomen.

"The heart is the organ which receives most relief, because by the time such abdominal distension has been reached, the ribs have become permanently pressed outwards, the outer attachments of the diaphragm are rasied, and the lungs do not receive much relief. While the heart is directly relieved from upward pressure, its work is farther lightened because the circulation is carried on more easily, the great vessels no longer suffer from pressure in their passage through the abdominal cavity, while organs receiving a large blood supply, such as the liver, spleen, and kidneys, have also a more free circulation. The heart is not only relieved from direct pressure, but the general circulation is more easily carried on. Hence, one frequently sees a poor woman with an

enormously distended abdomen, bright and active while on her feet, and appearing to suffer little from her condition; but keep her quiet or attempt to keep her in bed, and a change at once takes place. Her breathing becomes labored, her heart acts irregularly, her pulse becomes feeble and intermittent, her complexion is rendered more or less cyanotic from imperfect aeration of the blood, her kidneys and bowels act sluggishly. The excretory organs act imperfectly and fluid accumulates more rapidly, and the onward progress, to the much more serious indirect displacement of the heart, is hastened. Frequently, when first examining a patient in bed, the general symptoms above described, with irregular and intermittent pulse, make one fear cardiac mischief, but on seeing her up and moving about, one is surprised to find a perfectly regular pulse.

"The due appreciation of this condition is very important in practice, and it is not uncommon to find a patient much more distressed by her disease than she need be, because officious friends, or, perhaps, even an inexperienced medical attendant, keep her in bed or confined to the sofa, persuading her that it is dangerous to move about. A patient who has been judiciously managed in this respect, may be at once submitted to ovariotomy, even when the distension is great; whereas, one who has been made an invalid of must be first tapped, in order that the heart, lungs, etc., may be restored to healthy action before the ordeal of a great operation can be safely faced.

"Of course the healthy performance of the functions of the bowels, kidneys, etc., must be attended to, at the same time that the patient is advised to take gentle exercise, mild aperients; citrate of potash combined with a little quinine being the therapeutic agents of most service.

"If the distension has become so excessive that this stage is passed, or if from fixing of portions of the tumor in the pelvis œdema of the feet and legs has rendered exercise impossible, we shall very soon be called upon to consider the much more serious forms of indirect displacement of the heart. Indirect displacements of the heart may be met with along with ovarian disease or other abdominal tumors, owing to some lung affection or other malady existing with, but independent of, the abdominal condi-

tion, the latter not being sufficiently advanced to interfere with the action of the diaphragm ; such cases are, however, entirely outside our present consideration. Indirect displacement, due to great distension of the abdomen, or to more moderate distension combined with the pelvic condition named above, may be of several kinds, and the actual situation of the heart will vary according to the position of the displacing cause; but space will not permit a detailed description of such cases.

"Fluid may accumulate in one or other pleural cavity, or in both, and this may happen either as a part of a general effusion of fluid due to the interference with the circulation, or as the result of spread of malignant disease from the peritoneum, either in cases of cancer, or in those in which an ovarian cyst containing papillomata has been tapped or has ruptured, causing peritoneal infection. In cases in which fluid accumulates in both pleuræ, it is easy to see how seriously the heart's action may be interfered with, especially when it is already pressed out of place by upward pressure of the diaphragm. In such a case, the same causes which have led to accumulation of fluid in the pleuræ, will also cause its presence in the pericardium.

"These are the cases in which we hear of sudden death due to ovarian disease; œdema, or collapse of the lungs, coming on to put the final bar to the action of the heart—already displaced and hampered in every possible way.

"It is more common to find fluid in one pleura than in both, though it is not easy to explain why this should be.

"Fluid may be poured into the pleura so as to compress the lung and be reabsorbed ; the lung does not at once expand, and the heart, from being displaced to the one side, will now be found drawn over to the other, and this is a possibility which must not be overlooked in dealing with these cases. For should a patient be subjected to ovariotomy before the lung has recovered itself, the result may be disastrous. Such a case once occurred in my own hands, and I, therefore, give the warning on good grounds. It is a condition very apt to be overlooked or misunderstood, because the great distension from below partially fills up the vacuum, and the retraction of the chest-wall is not so well marked as in a case uncomplicated by abdominal distension. If the case has

been seen before the change occurred, the heart displacement will at once attract attention; but, if not, one is apt to regard the dulness as due to fluid in the pleura, a condition often relieved by ovariotomy. Whereas, if there is a lung which has failed to expand after the disappearance of the fluid in the pleura, the prognosis is much more unfavorable.

"The surgical treatment of these cases must vary according to their special features, but the following general rules may be laid down.

"If the patient is merely suffering from the direct displacement in its mildest form, and is well when moving about, other things being favorable, ovariotomy may be at once performed.

"If she has gone a little beyond this condition, and is suffering from symptoms which are the prelude to indirect displacements, it may be advisable to tap and wait for a time till the re-establishment of free circulation has caused a return to healthy action in kidneys, lungs, etc. But in most cases, especially now we have antiseptics to aid us, ovariotomy may be safely performed at once, even at this stage.

"If some amount of fluid be present in one or both pleuræ, or if œdema of the abdominal parietes and legs be marked, ovariotomy may still be safely performed, if the heart has been previously healthy. Tapping is, at best, the adding of another danger and more suffering, and should always be avoided if possible.

"If the surgeon be called upon, however, to treat a case already *in extremis*, with some of the worst forms of indirect heart displacement, and a large accumulation of fluid in a cyst or in the peritoneum, ovariotomy is out of the question, or too dangerous to be attempted; and instant tapping is indicated. Sometimes it may be advisable to tap the pleura at the same time. Even in cases of malignant disease the patient's sufferings may often be much relieved by a tapping, though the end is often hastened by the exhaustion caused by rapid reaccumulation of fluid."

The practical remarks on the relief of the heart displacement will apply to the treatment of displacement, due to accumulations of fluid, not due to ovarian disease.

Strain and Shock.—Though scarcely quite in place here, these

maladies may probably be discussed more appropriately in this than in any other chapter.

Strain is a comparatively common condition, and I have found it in runners, in athletes, football players, cricketers, and oarsmen. It is, of course, more readily induced in persons with small hearts than in other persons. The effects of overexertion systematically pursued, are, overdistension of the right side of the heart, and diminution of capacity to maintain effort for long. Thus, swimmers talk of "training on," so long as they can dive for lengthened and longer periods, and of "training off" when their capacity is diminishing. In the first case, the efforts are within the capacity for development on the part of the right chamber of the heart; the latter when it is being exceeded, and instead of becoming more equal to the stress laid upon it, it grows less equal thereto, showing that the demand upon it is too great. A well-marked case is given by Dr. Clifford Allbutt in his essay on the Effects of Overwork and Strain in the Heart and Great Bloodvessels. The man was an out-patient of the Leeds Infirmary, and Dr. Allbutt diagnosed the case as one of "overstrained heart." At his request I drew up the following notes for him:

"He is a well-made active fellow, æt. 29. He lives two miles from his work and often runs the whole distance, as he adds the profession of runner to that of dyer. He often runs to Bradford and back, and is thus constantly overtaxing the pulmonary circulation and right ventricle. The physical signs are: venous pulsation and excited action of the heart, especially of the right ventricle. No murmur, pulmonary second sound accentuated; dulness extends from below the fifth rib into the epigastrium and to the right of the sternum. When holding his breath pulsation is very visible below the ensiform cartilage; pulse 84, compressible. On exertion a slight irregularity of the heart is set up. Symptoms, general health good, but is not well fed, his wages being low. He is now soon out of wind, and suffers from palpitation. He does not smoke much. Diagnosis, distension of the right ventricle and auricle from long overtaxation."

I remember very distinctly this man saying, in answer to several questions, that often he allowed himself too little time for his run to the works, and that, consequently, he had to make greater

efforts than he would otherwise have made, in order to get in before the yard bell ceased ringing, otherwise he would have lost so much time.

The symptoms generally presented by persons with overstrained hearts are as given above. The marked symptom is that they are out of breath readily, and can no longer make the efforts of which they were formerly capable. Palpitation is easily induced, which was not the case formerly. A friend of mine, a stalwart man of forty-six, with a hypertrophied heart and tense vessels, ran one day up the steps and along the platform of a railway station, just succeeding in catching the last carriage as the train was moving away. He did not suffer much discomfort at the time, but the effects of what he had done were readily demonstrated by palpitation and dyspnœa, showing themselves on slight effort; and months elapsed before he could run any distance again.

The same overstrain of the heart is found in horses who have been over-galloped, and become "broken-winded," so called. This condition is one of overdistension of the right heart; and is remedied by the animal being turned out to grass, where his exertions are limited by his sensations and not by his rider's wishes. This differs from the vesicula emphysema of chronic broken-windedness.

By the courtesy of Mr. Moore, the well-known veterinarian, I have had the opportunity afforded me of seeing a number of horses with this cardiac form of broken-windedness. The heart beat somewhat feebly and irregularly in each case.

Mr. Myers, in his Alexandra Prize Essay, has pointed out how the tightly-fitting accoutrements of the soldier expose him to cardiac derangement; and Dr. Arthur Davy has described how the strained position of the recruit at drill tells upon his circulatory organs. The chest is fully dilated, and with this condition of the chest the drill is carried out. He advocates a more natural form of drill as a means of lessening the heart disease so notoriously prevalent in the army.

In addition to excitability of the heart's action and palpitation readily induced, there may be found a powerful impulse and a loud first sound in cases of overstrained hearts, where natural attempts at muscular compensatory hypertrophy have been made,

but have not been perfectly successful. Probably where perfect muscular compensation has been attained, we do not see the case till aortic valvulitis has been developed (p. 110). Strain on the heart may lead to rupture of an aortic cusp, and, according to Clifford Allbutt, of a mitral velum also.

The incapacity induced by overstrain of the heart and right-side dilatation, which cripples a whilome able athlete, must be distinguished from the limited capacity of persons with congenitally small hearts, described by Latham as follows: "Many people have an habitually feeble circulation, and an occasionally irregular or intermitting pulse all their lives. Yet they pass for well, and really are well in their own consciousness, and live as long as other people. In several such I have convinced myself (as far as it is possible to be convinced about such a matter) that their heart has been weak of structure, and of small size in proportion to their stature; and it is a curious fact that most of those in whom I have found this condition of heart, they have told me that they were always short-winded, and that at school they could not exert themselves like other boys; they were accounted bad runners."

As to the treatment of this condition of strain it is obvious the principles laid down in this chapter on treatment must be strictly applied. All exertion must be abandoned, athletics laid aside, a quiet existence maintained; the nutrition must be kept up, and tone given to the heart by the administration of digitalis, strychnia, and iron; while the excitability will be calmed, and the palpitation relieved by a belladonna plaster. I have no experience of the German plan of wearing a flask of iced water over the heart. This may give relief, but at best it can be but a palliative, and not a curative, measure.

Shock, as a cause of actual disease of the heart, is a matter of which I have little personal experience, consequently I shall place before the reader an abstract of Latham's interesting chapter on the subject. A stableman was running a horse, showing him for sale. Immediately afterwards severe palpitation showed itself; his lips became blue, his breath short, and his left side painful. This continued for twelve months. After ten weeks, when his cough and dyspnœa were peculiarly distressing, he came into hospital. His heart was much enlarged, there was a blowing

systolic sound at the aortic valves, and the second sound was indistinct. His legs swelled, and in seven weeks he died. The heart was enormously enlarged chiefly by dilatation, and there was rupture of an aortic cusp. In another case acute endocarditis followed a severe effort at rowing, leaving behind it mitral disease and hypertrophy, with dilatation of the left ventricle.

A young gentleman, who had lived fast, in the midst of vigorous rowing was seized with sudden pain at the heart, excessive impulse, and sense of approaching death. In this case there was no valvular disease set up, but there was some hypertrophy. Palpitation is the resultant trouble, and once or twice has been excessive, and accompanied by a death-like feeling. "Whatever he does, business and pleasure, eating and drinking, are all under the restraint of continual watchfulness, for the sake of moderating the palpitation of his heart."

In another case a young officer, who had also lived fast, and had had occasional palpitation, after dancing all night, plunged into a cold bath and went on parade. Violent palpitation came on, and he was incapacitated from duty from that hour. A fortnight later a very severe attack of palpitation came on, his lips became blue, and he experienced the sensation of impending death. He was freely bled. Months afterwards his heart was a source of the utmost anxiety to him.

A young gentleman, running quickly, came in contact with another person running in an opposite direction. He crawled home, and for many months was seriously ill. He had pain in the heart, with excessive impulse. After the lapse of many months he returned to business, having lost the constant palpitation, but liable to returns of it upon occasions of excitement. At length he lost it and led an active life for five-and-twenty years. A man one night was wofully maltreated and robbed. He lay all night insensible, and next day was found and taken home. He had before that been perfectly well, but palpitation and dyspnœa from that time prevented him from ever again returning to work. Some months afterwards he went into St. Bartholomew's with dropsy and the evidence of hypertrophy with dilatation. "He soon died, and his heart was found of a size which was almost incredible. All its muscular structure was

enormously amplified, and all its cavities enormously dilated, its pericardium and lining membrane and valves free from disease." In another case a similar condition of the heart resulted from a transport of rage.

Our knowledge of the effects of shock upon the heart, except in the case of rupture of the aortic valves, is very scanty, but there seems evidence enough here to warrant the hypothesis that the nutrition of the heart may be profoundly influenced by shocks, not necessarily bodily. Latham says that "the ultimate result is a good deal according to the treatment which the patient meets with at the time." Now bleeding was in vogue at the time of these cases, and of course the question suggests itself, how far the bleeding had a baneful effect upon the different cases? The whole subject is one of the deepest interest, but at the present time it is shrouded in obscurity.

Seitz has written on Strain ("Die Ueberanstrengung des Herzens"), and in it has translated the essays of Allbutt, Myers, and Da Costa, to which he has added a number of cases of his own which are worth studying, as he has followed them to their fatal termination.

I have seen several cases of overstrain of the heart, with a mitral systolic murmur, in boys who have been compelled to run long distances, as in pursuit of a pleasure-van. In time the heart becomes quite normal under digitalis and iron.

CHAPTER XI.

ANGINA PECTORIS—SEVERAL FORMS—SUDDEN DEATH.

It has appeared desirable, for several reasons, to consider angina pectoris and sudden death together in one chapter. By such means the cause of sudden stoppage of the heart will be best elucidated. Still even this plan will not clear away the difficulties which yet surround the pathology of angina pectoris. It is a disease of paroxysmal nature, having, usually, intervals when the health is apparently perfect. Heberden classed it amidst the *distensiones* or spasms. Its paroxysmal nature is now, as it has always been, recognized; but the view now obtaining is that it is a state of acute distension of the heart, not a state of spasm of it. The remark, therefore, of W. T. Gairdner, that "the question, as between spasm and paralysis, therefore, is one of great difficulty, if not, indeed, practically insoluble in the present state of our knowledge," scarcely applies now.

In proceeding to consider angina pectoris, the feeling is strong to go into the literature of this subject, and the temptation to do so is great in consequence of its deeply interesting nature. But the intention of this work forbids this, and the temptation must be resisted. Reference, however, to the excellent chapter on this subject by the late Dr. Latham, in his lectures, by W. T. Gairdner in Reynolds's "System of Medicine," and by Eulenburg in Ziemmsen's "Encyclopædia," must be made. Further, readers possessing more than an ordinary interest in angina will find in these articles much that will repay them for the time spent in their perusal.

Angina may be divided into true angina (vasomotor), and false or neuralgic angina. True angina may briefly be said to be due to vasomotor spasm of the small vessels—a rise in blood pressure in the arteries and a condition of acute distension of the left chamber of the heart. Consequently, angina is found under

circumstances where palpitation is common. Where there is palpitation the heart is struggling actively against the obstruction to be overcome on its systole; where there is angina the distension is great and the heart is almost paralyzed. Neuralgic or false angina is a malady found under other circumstances, as in women at the menopause, and is not so distinctly connected with arteriole spasm. Latham says truly enough of angina, "We are sure of what it is as an assemblage of symptoms. We are not sure of what it is as a disease."

As to the symptoms of angina pectoris, the two prominent subjective phenomena are pain in the chest and a sense of impending death. The pain is of an unusually trying form, such as to task the greatest fortitude. Arnold, of Rugby, said he "could scarcely bear it if it were as severe as it had been." But it is not the severity of the pain alone which gives it its terrible feeling of menace; it is the sense of impending death which accompanies it that renders it so intolerable.

The aspect of the patient is indicative of the agony which he endures. Sweat gathers on the brow, or drops off quickly, the face is pallid and bloodless; the patient feels as if to move were to court instant dissolution; the breathing is shallow, there is no gasping for breath, no labored respiration. The pulse at the wrist may be small, compressible, regular or irregular, or tight and wiry. The pain shoots through the chest at some point of the sternum and down the left arm, and even out at the little finger. In one of the most severe cases I have ever seen the pain shot out at each side of the occiput and from the spines of the shoulderblades, as well as down the left arm and fingers. Dr. Heberden said of this "breast-pang:" "(1.) It comes suddenly and goes suddenly. (2.) It has long and complete intermissions. (3.) Wine and spirituous drinks and opium afford great relief. (4.) It is increased by mental agitation. (5.) It exists for years without other injury of the health. (6.) At first it is not excited by exercise in a carriage or on horseback, as is usually the case with scirrhus or inflammation (organic disease). (7.) The pulse is not quickened in the very paroxysm. (8.) The paroxysm attacks some after their first sleep; a frequent event in diseases which arise from spasm." It may or may not be associated with dis-

ease of the heart, but it very commonly is, and in fatal cases some change in the nutrition of the heart has been invariably found.

Now what are the associations of angina pectoris? Probably these will tell us much as to the nature of the malady. "Seldom at first can it be imputed to any obvious cause but undue bodily exertion. And for a considerable period (perhaps for months, perhaps for years), seldom can it be imputed to anything beside this undue bodily exertion and sudden or strong agitation" (Latham). In going over the list of cases given by Latham, in most there is a history of effort badly borne as well as attacks of angina. In Dr. Arnold's case alone there were no prodromata of the sudden change impending. Yet even here morbid changes were found. "The heart was rather large. It was very flaccid and fat in its appearance. All the valves were quite healthy; the orifices of all the great vessels were quite natural. The muscular structure of the heart in every part was remarkably thin, soft, and loose in its texture. The walls of the right ventricle were especially thin, in some parts not much thicker than the aorta, and very loose and flabby in their texture. Its cavity was large. The walls of the left ventricle, too, were much thinner and softer than natural. And the muscular fibres of the heart generally were pale and brown. The aorta was of a brown-red color throughout its internal surface, probably from putrefaction. A few slight atheromatous deposits were observed in the descending aorta. There was but one coronary artery, and, considering the size of the heart, it appeared to be of small dimensions. It with some difficulty admitted a small director. It was slit open to the extent of nearly three inches. Its internal surface was red but healthy, with the exception of a slight atheromatous deposit situate about an inch from the orifice of the artery." Some commencing imperfect nutrition of the heart-walls must be suspected here. Latham sums up his thirteen cases so. The impulse in some cases was very feeble, and there was much resonance over the præcordial region. In others the area of dulness was great, "the bulk of the organ was augmented withal; augmented, however, by the addition of something which was a source of weakness, most probably of fat." In others, murmurs were heard indicating disease of the aortic valves.

W. T. Gairdner, like Latham, feels the difficulties before us "in this difficult inquiry in which we are reduced to the study of 'broken lights' and fragments of truth." He gives the case of Seneca as possibly one of angina pectoris. In speaking of the case of Dr. Chalmers, he says: "In very few of those cases in which the fatal result has been most sudden and startling, have there been any such records of the incidents preceding death as are given here. In not a few of the cases observed personally by, or more or less intimately known to the author of this article, there has been reason to believe that considerable suffering, or sense of disability, though not always of one and the same character, has been present; and in some of these it might easily, perhaps, have escaped attention had the individual been extremely reticent, or not surrounded by anxious friends intent upon everything that appeared to affect the comfort of one dear to them." Dr. Chalmers had been undergoing considerable exertion during the last month of his life. On the Sunday he did not get up to breakfast, but attended church, walked some distance with a friend homewards, spent the evening in apparent good health and spirits; "wrote his sister a hopeful and affectionate letter, expressive of perfect contentment and satisfaction. He retired to rest at the usual time, and next morning was discovered dead and cold. The body had an attitude of calm repose. The bedclothes were scarcely disordered; on them rested a basin which had received the contents of the stomach. This was the only evidence of anything like a death-struggle."

The account of the great John Hunter is succinctly given by Dr. Gairdner. In 1773, twenty years before his death, he had his first attack of angina pectoris in his forty-fifth year. Three years later he had a second attack; he had no third attack till 1785. From this time onwards he became increasingly subject to paroxysmal attacks, which assumed more and more the characters of typical angina pectoris. In the most severe attacks he sunk into a swoon or doze which lasted about ten minutes, after which he started up without the least recollection of what had passed or of his preceding illness. The agonies were dreadful, and when he fainted away he was thought to be dead. Yet he ate and slept as well as ever, and his mind was in no degree de-

pressed; the want of exercise made him grow unusually fat. His disease was called "flying gout," though Ed. Jenner diagnosed it as angina pectoris. He was deeply sensible of the risk to which he was sometimes exposed by overexertion, and still more by his uncontrollable temper; he was accustomed to say that "his life was in the hands of any rascal who chose to annoy and tease him," a remarkable expression, and a sad anticipation of the actual ending. Very much impaired in health at last, on October 16th, 1793, he determined to be present at a meeting at St. George's Hospital. Something he said in the Board-room was flatly contradicted, he left the room in a silent rage, had just gained the next room, gave a deep groan and fell down dead. The appearances in the dead body were complex. The pericardium was very unusually thickened; the heart very small, its muscular substance pale; the coronary arteries were converted into open bony tubes; the valves of the left side of the heart also were involved in a similar degeneration; the aorta was dilated, in its ascending part, to the extent of one-third. The carotid and vertebral arteries within the cranium were also bony, and the basilar artery "had opaque white spots very generally along its coats." The structure of the brain itself was normal.

From such surroundings it is clear that fatal angina pectoris is found when the muscular tissue of the heart is undermined by impaired nutrition, and especially when this is associated with ossification of the coronary arteries.

It is now time to inquire into the pathology of angina pectoris. That the breast-pang is connected with a condition of the heart itself is not in dispute. But that its causal associations are to be sought for elsewhere is almost, if not equally, certain. Latham says: "But beyond the heart, the aorta, and its branches within the chest, we must look to the vascular system everywhere, when we have to do with a case of angina pectoris." The knowledge of that time did not permit of Dr. Latham's detecting the commencing-point, else he surely would have unravelled the mystery. The vasomotor spasm, which is now known to be the starting-point of most cases of angina, was not then comprehended or comprehensible. "We now know that the typical angina is only the culminating form of a group of symptoms which in their less pro-

nounced, less definitely painful, and more complicated form, are found to penetrate the whole field of cardiac pathology and diagnosis. The angina which consists purely of a paroxysm of pain, and of a paroxysm which kills suddenly and instantaneously, is rare; but the angina which consists of a tendency to paroxysmal aggravations (not always purely of pain) superinduced upon, and complicating, the other symptoms and sequelæ of cardiac organic diseases, is a matter of every-day experience." W. T. Gairdner is quite in accordance with my own experience in this expression of opinion; and imperfect anginal attacks are comparatively common, especially in the subjects of disease of the aortic valves.

That angina pectoris is associated with vasomotor spasm is now scarcely disputed.

In a case of aortic disease in the Royal Infirmary in Edinburgh, in December, 1866, there were palpitation and throbbing of the carotids as well as severe angina. Dr. Lauder Brunton made repeated sphygmographic tracings of the radial pulse, and found that there was increased tension in the arteries during the attack, which he attributed to contraction of the small systemic vessels, so sudden and so great as to deserve the name of spasmodic. Knowing the effects of amyl nitrite upon the small arteries and arterioles, as observed by B. W. Richardson and A. Gamgee, he administered it by inhalation; the arterial pressure was lowered and the attacks relieved. This was repeatedly done, and the relations of increased arterial tension to angina-spasm thoroughly established. Further, small bloodlettings, to relieve the arterial tension, were found to exercise a notable influence. Digitalis made the pain worse. The extended use of amyl nitrite in the relief of angina has corroborated the views of Dr. Brunton as to the cause of anginal attacks. If there were temporary palsy of the heart the blood pressure in the arteries would fall. But the high arterial tension shows that the outflow through the peripheral vessels is obstructed, and then the left ventricle contracts with difficulty; it is imperfectly emptied at each systole, it becomes acutely distended, and breast-pang is the consequence. Palpitation is often induced by the same cause; and palpitation is found in some cases where with similar associations angina is found in other persons.

The observations of Brunton do not stand alone. Eichwald observed that in some cases of severe hysteria there were steno-cardiac attacks, with a rise in arterial tension; and the fact that the arteries are corded in an attack of hysteria, and that a large bulk of pale urine—the associate of high arterial tension—is passed after the attack, are well-known clinical facts.

Traube held that the diminished volume of the arteries, with increased tension in them often found with steno-cardia, was related to an increased stimulation of the nerve centre of the vaso-motor system. Cahen thought angina pectoris connected with the vasomotor nerves. Landois viewed some cases of excessive nervous palpitation as being related to vasomotor angina. Nothnagel has worked at the subject very thoroughly. He has shown that a sudden check to the circulation in the extremities, which is induced by spasm of vasomotor origin, is the cause of increased action of the heart, palpitation and angina (W. T. Gairdner says "pseudo-angina"). The whole subject is being cleared up rapidly. Imperfect anginal attacks are much more common than fully developed attacks, and are found in persons of gouty diathesis. They occur comparatively early in life in men whose fathers have died of angina. One such case I know well in a gentleman of literary habits. He had attacks of recurring angina, for which he saw me in September, 1874; the attacks came on frequently and were accompanied by a fear of death. His aortic second sound was accentuated, his pulse tense, and his heart's action rather hammering. He had been eating meat extensively under the impression that such a dietary would strengthen him. The amount of meat in his dietary was reduced, and he had a mixture of potash and buchu. In three weeks he was much better, the attacks had left him, and he was feeling generally better. He has continued well since; whenever he feels a threatening of angina he takes the mixture and is soon all right. While the heart textures are sound, attacks of angina are little fraught with danger; but when the heart-walls become degenerate the danger of the heart stopping in diastole from acute distension becomes great. This man may, probably will, die years hence of angina as his father did.

We know, as we shall see at length in the chapter on the gouty heart, that persisting arteriole contraction leads to hypertrophy of

the left ventricle; sudden spasm leads to sudden increase of arterial tension and palpitation; sudden arteriole spasm, if severe, may lead to stoppage of the heart in diastole; this is the angina which kills. When the patient recovers the heart has not stopped, but has been acutely overdistended, and of this acute distension agonizing breast-pang is the pathognomonic indication. Conditions of the coronary circulation may add to the heart's embarrassment. V. Bezold has found that feeble and irregular action of the heart with ultimate cessation follows ligature of the coronary vessels. W. T. Gairdner writes: "Now, apart from the obvious bearings of these facts upon the case of organic obstruction or constriction of the coronary vessels (perhaps the most clearly established of all the permanent organic changes in connection with fatal angina pectoris), is it not extremely probable that a similar effect, or an aggravation of a pre-existing tendency to interrupted cardiac action might occur if, in a case of disease of the aorta or coronary arteries, cardiac anæmia were aggravated for the moment by vasomotor spasm of the smaller arteries within the heart itself?"

It is possible to erect the hypothesis that the presence of spasm of the coronary vessels makes the difference betwixt attacks of palpitation and attacks of angina pectoris. When there is a sudden rise in the blood pressure in the arteries, due to vasomotor spasm of the peripheral systemic arterioles, and the heart-walls are strong and well nourished, palpitation is evoked; when the coronary branches are involved in the vasomotor spasm then angina is produced, and the heart-walls, acutely distended with the blood, can scarcely contract in the face of the opposition presented to their contraction by the high arterial blood pressure. When this sudden systemic arteriole spasm extends to the coronary vessel in a heart whose walls are diseased, a fatal attack of angina with the heart full of blood in diastole may be induced. The danger increases with the extent of the structural degeneration of the heart-walls. Sudden rises of blood pressure in the arteries will tax hearts in their textural integrity, and lead to painful distension; such sudden demands on decayed hearts lead to agonizing angina pectoris, and the sense of impending dissolution is frequently followed by sudden death.

From this it will be seen that true angina pectoris is part of a morbid process, not a disease of the heart *per se*. It is most commonly found in persons whose vasomotor system is disturbed, namely, the subjects of lithiasis. In the early stages of lithiasis the attacks are scarcely serious; in advanced condition of degeneration of the tissues of the vascular system angina pectoris is fraught with imminent danger. Some cases go on for years, as in John Hunter's case, the increasing disease of the vascular system adding to the danger in each recurring attack, until at last the fatal attack comes. Arnold died of a first attack; a patient of mine died in his second attack. Forgetful of the counsel given him after the first attack, he indiscreetly overexerted himself and brought on a second, which was almost instantly fatal. The prognosis then varies with the associations and the condition of the heart-walls. Where atheromatous arteries tell of degenerative changes then the prognosis of angina pectoris becomes very grave. As to the actual cause of death W. T. Gairdner sums up: "It must, however, be conceded to the advocates of the theory of paralysis, pure and simple, that nothing but the presence of severe pain in the angina-paroxysm, and the absence of this symptom, as a rule, in purely paralytic affections, tends to support the spasm-theory of angina. Post-mortem examinations have generally shown that the heart is found flaccid, rather than rigidly contracted; and the lesions found in the muscular substance of the heart itself are usually such as would confirm the idea of decidedly and permanently weakened energy, rather than a disposition to abnormal contraction." The pain is that of overdistension in all probability, and thus the last support of the spasm-theory falls to the ground. The pain is probably analogous to that felt in voluntary muscles when overexcited (Eichwald).

The treatment of angina consists of two distinct lines, viz., the treatment of the interparoxysmal stage, and the treatment of the paroxysm itself. Pure angina pectoris, *i. e.*, the form due to arteriole spasm (Nothnagel's Angina Pectoris Vasomotoria), is now solely being considered. Its association with lithiasis, and all the morbid outcomes thereof, indicates a non-gouty dietary. Brown meats should be very sparingly taken, and the dietary

which will be given under the treatment of the gouty heart should be strictly adhered to. Then it is well to remember that in the form of uric acid and sparingly soluble urates, do the products of albuminoids in their retrograde career, linger in the system in cases of imperfect blood depuration. Potash with buchu is the remedy *par excellence*. In one of the worst cases of angina I have ever seen this plan of treatment gave great relief. This patient, a gentleman of forty-two, who had lived well, had begun to suffer from recurring attacks of angina, which were readily induced by exercise. He could not say how many attacks he might have had. There was hypertrophy of the left ventricle with a loud aortic second sound, and a faint systolic whiff heard at the base of the heart at intervals. The urine contained deposits ; and he felt a good deal of irritability at times. The pain in the attacks shot out at the occiput and shoulder-blades as well as the finger-ends, when the attack was severe. The patient also had *tic convulsif*, or blepharospasm on one side of the face. Some difficulty was experienced in determining the nature of the angina. The condition of the heart gave the malady a gouty aspect. The twitching of the muscles made it look neurosal. The medical gentleman who brought the patient had observed that the pulse became hard and incompressible during the attack, resuming its normal form as the pain vanished. This settled the question of arteriole spasm. The patient was put on a non-nitrogenized dietary, and recommended to keep quiet. He was ordered some bicarbonate of potash, with small doses of strychnia and digitalis. The attacks passed away gradually, the pulse became softer, and the heart sounds quieter. The patient felt much relieved. The tic was also improved. Probably it also was of gouty origin. Unfortunately it has not been possible to procure an account of the future history and present condition of this patient.

Then comes the treatment of the attack itself. The claims of nitrite of amyl to our confidence have been substantiated by other cases, as well as the remarkable pioneer case of Dr. Lauder Brunton's. Dr. Herries Madden, of Torquay, has related, in the "Practitioner" for 1872, his own experience. He began to suffer from severe angina in 1872, and after trying several reme-

dies, found much relief from inhalations of amyl nitrite. The attack instead of lasting twenty minutes was cut down to two, strangled as it were. The attacks first became less severe, then less frequent, and finally ceased. In 1875, he had not required its use for a considerable time. In December, 1878, Dr. Madden kindly writes me in answer to an inquiry, "My health continues good, and I am able to carry on my work without difficulty. Anginal attacks recur occasionally but not with any severity. It is a long time since I have needed to use the amyl. On one point I have to be particularly careful—I dare not hurry myself in anything. If I do, through inadvertence, I am speedily reminded of my mistake by an attack of pain—more or less sharp."

The amyl nitrite is readily carried about in a small bottle with a piece of lint, and seven or ten drops may be inhaled when the attack comes on. There seems every reason to suppose that attacks of hysteria accompanied by palpitation would be equally effectually removed by amyl inhalations.

Heberden advised cordials with full doses of opium, but such is not the practice now in vogue. Dr. Gairdner advocates warm mustard pediluvia, with heat applied to the arms and thorax. Such treatment is sound, for it meets the two great indications—to dilate the peripheral capillaries and to stimulate the heart, which heat applied locally to the thoracic wall does. Hydrate of chloral is a very doubtful remedy to apply, from its tendency to paralyze the circulatory as well as the respiratory centres. Eulenburg has employed the constant current with success. Digitalis gave decided relief in two cases, which were, however, both of the neurosal form in women at the menopause, one of whom had a dilated heart. It is quite permissible, however, as in the case given above, to administer digitalis with or without nux vomica, along with full doses of potash, when the heart is seriously embarrassed in the attacks. It may be necessary to excite more energetic contractions of the heart while removing the cause of the vasomotor spasm, viz., the waste nitrogenized material in the system.

Angina pectoris, however, is not always of vasomotor origin; though this is its common origin. Before proceeding to consider

the other forms, some further testimony as to the vasomotor origin may be tendered. Eulenburg, who classes angina pectoris among the vasomotor neuroses, says, "During the attack the skin is often pale, cold, dry, almost bloodless in the hands and feet, the face pale and sunken; a general sensation of chill, chattering of the teeth, paralgic sensations (formication and prickling) in the tips of the fingers, may be caused by the cutaneous anæmia. Towards the close of the attack, or after it, there may be the contrary condition of redness, swelling, increased sense of warmth in the skin, and abundant perspiration may be secreted." He quotes a case of Heubner's, where the attacks first appeared in a robust man of seventy-four after a long ride in a post-wagon in cold weather. Such exposure produces contraction of the whole of the cutaneous capillaries, and so drives the blood inwardly; this action is requisite to the maintenance of the body temperature, for by it the mass of blood in the external or heat-losing area of Rosenthal is lessened, and, consequently, the bulk of blood in the internal or heat-producing area is increased. But this sudden increase of blood in certain parts tests the integrity of the vascular tunics; if the arteries are decayed apoplexy is the result; if the heart-walls are unsound cessation of the heart in diastole is the consequence. When cold fits of weather set in quickly, cases of sudden death are quite common. A number of notable persons died either of apoplexy or heart failure during a spell of sudden cold in May, 1878. M. Thiers died after exposure to cold. Males are much more liable to vasomotor angina than females; this is due to their being more frequently the subject of lithiasis. All the subjects of lithiasis, with its morbid sequelæ in the vascular system, should be carefully warned to clothe themselves in extra flannels when the temperature falls, especially when going out from warm rooms.

The pain of angina is distinctly located in or about the midsternum, whence it radiates. Eulenburg says this is due to the connections between the superior cardiac nerve, and the anterior branches of the four upper cervical nerves; while the middle and inferior cardiac nerves are connected with the four lower cervical nerves uniting in the brachial plexus and first dorsal nerve. The pain usually runs out at the peripheral endings of the ulnar nerve,

especially the little finger. It is almost invariably found on the left side only. In a case where it was on the right side the pulmonary artery was the seat of disease.—"Pathological Soc. Trans.," 1878.

Landois described several varieties of angina. In addition to the vasomotor form he gives other three: (1) disturbance of the excitomotor cardiac nervous system; (2) conditions of irritation of the cardiac branches of the vagus; and (3) reflex excitement due to irritation in the abdominal organs. We know well that palpitation may be due to disturbance in the abdominal viscera, and still more in the pelvic viscera. We know that collapse and syncope follow blows upon the testicle, and that sudden death is frequently the result of a blow upon the pit of the stomach. Prostration and collapse are produced by irritant poisons. How in these cases the heart and circulation is affected, we do not yet know. The most probable explanation is that the effects are produced by means of the reflex inhibitory nerves of the heart. By producing syncope and unconsciousness the brain is anæsthetized, when a sudden shock, emotional or physical, is experienced. But the mechanism which secures this may be the means by which angina may be set up under certain circumstances. A stone in the kidney produces vomiting; a tender ovary may produce vomiting, palpitation, or a cough; just as pregnancy sets up vomiting in the early months, or a cough, called in Scotland "a cradle cough." So we can understand how anginal attacks may originate in some disturbance in a viscus. Of the forms due to vasomotor spasm and to irritation (direct) of the vagus, we know something of their pathology; of the other forms of angina little positively yet, unless it be mere speculation. Eulenburg truthfully says, "Pathological anatomy has, as yet, contributed little that is of use; angina pectoris belongs to that class of neuroses which show most convincingly how much we owe to the discoveries of experimental physiology and their pathological application, and how far mistaken in point of fact those are who expect to find in pathological anatomy the source of every improvement in nervous pathology. Perhaps no ray of light would ever have penetrated the obscurity in which this disease is wrapped, if physi-

ology had not shot forth those lightning flashes of hers, which threw such a wonderful illumination upon the whole matter."

The neurosal disturbance sometimes involves the respiration, and W. T. Gairdner says, "This peculiar type of 'suspirious' or irregular sighing respiration is so far characteristic of the 'angina *sine dolore*,' that I cannot but regard it as being in some way related to lesions involving the respiration through the cardiac nerves." Stokes says of this form of breathing, "It is, when confirmed, almost pathognomonic of a weak, and, in all probability, a fatty heart; but whether it is to be taken as indicative of the predominance of the fatty change being on the right side of the heart, is still an open question. In well-marked cases the respiration is shallow and not labored. In attacks of cardiac dyspnœa, which have been mistakenly supposed to be attacks of angina by those who are not familiar with the subject, the respiration is labored; the lungs are gorged with blood, the breathing-space is diminished; while the heart palpitates from the demand on the right chambers. In angina pectoris there is nothing of this, rather an apnœal condition is approached."

Imperfect attacks of angina (vasomotor) are infinitely commoner than complete attacks. They are usually seen in persons of the nervous diathesis and gouty family history. Of little or no seriousness at forty, they are fraught with danger when the heart and arteries are rotten at seventy, or even at sixty-three.

Angina pectoris, due to irritation of the vagus through the inhibitory fibres, is a well-known form of this disease. Romberg, in his work on the "Diseases of the Nervous System," relates, under the heading of cardiac paralysis, a very remarkable case which occurred in Heine's ward in Vienna. A man, thirty-six years of age, complained of nervous symptoms and of his heart standing still. It intermitted for five or six beats. "The aspect of the patient showed that something terrible was going on within him; he sat as if thunderstruck (attonitus), speechless, motionless, his eyes wide open, his consciousness unimpaired. When asked about his sensations, he stated positively, that for a second or often more, he had a presentiment of coming arrest, in the shape of internal restlessness and oppression; that when the stoppage

took place, a violent pain seized both sides of the thorax, extending to the neck and then passing up to the nape and the head; that the pain remained fixed in the latter for some time after the attack, and that when the attacks were frequent, he could scarcely get rid of the sense of weight at the cervix." Attacks were brought on by emotion. During the intervals the heart presented no abnormal action and was quite healthy. The attacks increased in severity, and he died in a state of torpor. Heine called in Skoda and Rokitansky, and a tumor of the cardiac nerves was diagnosed. The cardiacus magnus nerve was woven into a black knot the size of a hazel-nut; the left vagus was involved in an underlying nodulated dark-blue lymphatic gland. The phrenic nerve was also embraced in a diseased gland. Irritation of the inhibitory fibres of the vagus was the cause of the long intermissions in this case.

Stoppage of the heart is also attainable by irritation of the sympathetic fibres in the abdomen, the *angina pectoris reflectoria* of Landois; and Bergson has related a case where anginal attacks ceased after treatment directed to the liver, which was enlarged. Cases are recorded in older literature of disease of the abdominal organs being accompanied by attacks of angina.

How far certain anginal attacks may be produced by abdominal irritation—as palpitation is undoubtedly so set up—we can only surmise.

False or neuralgic angina is seen chiefly in women, and the attacks are often severe. Some years ago I had repeated opportunities of witnessing terrible attacks of angina in a woman. She was nailed to the place where she was when the attack seized her, her face became pale, and beads of sweat stood on her brow. She could not bear to be disturbed for the purpose of examining her heart, but the pulse was very small. The attacks lasted some time, and were relieved by digitalis and alcohol. On a course of arsenic and digitalis the attacks disappeared. The woman was of very excitable temperament, with a violent temper, and was at the menopause at the time the attacks developed themselves. The attacks disappeared entirely, and after a lapse of twelve years have never reappeared; this indicates that they were not of vaso-motorial origin. In another case anginose attacks appeared in a

delicate woman with a dilated heart, who was also liable to severe attacks of spasmodic asthma. She was much benefited by digitalis. Eleven years have elapsed since she was my patient. She is still living. Where the angina is a neuralgia, as Laennec, or a hyperæsthesia of the cardiac plexus, as Romberg and Friedreich hold it, or an epileptiform neuralgia, as Trousseau termed it, then tonics, as arsenic and iron, or digitalis are indicated. In all future cases I shall carefully look for sources of irritation in other viscera, and especially the pelvic organs.

Sudden death from heart disease is probably due in all cases to failure of the heart in diastole. Spasmodic closure of the heart may occur, but it must be very rare, except when toxically induced. In aortic regurgitation sudden death is comparatively common, from the left ventricle being distended in diastole by the regurgitating current, and failing to contract. This is liable to occur when the nutrition of the heart is failing, probably in the cardiac ganglia as well as the muscular fibrillæ. Then the heart often ceases in sleep, but what is the cause of such failure we cannot yet say. When death is caused by shock, physical or emotional, we are compelled to suppose that the inhibitory fibres are thrown into action and fatal syncope follows. Fatal syncope differs from non-fatal syncope, probably, only in degree. When the system is exposed to shock the heart fails, and unconsciousness comes on from acute cerebral anæmia, and so the brain is anæsthetized for the time. Probably the anæsthetic effects of syncope lessen the shock to the brain at the time; but the severity of the shock, emotional or other, may be such as to arrest the heart's action permanently. We can scarcely suppose the heart's action to be arrested by a primary action on the cardiac ganglia.

CHAPTER XII.

THE TREATMENT OF ORGANIC DISEASE OF THE HEART.

The previous consideration of disease of the heart must point the lines on which the treatment must be conducted to be of any practical value. "The treatment of diseases rightly considered is, in fact, a part of their pathology," wrote the late Peter Mere Latham, and this is specially applicable to the diseases of the heart. In an organ acting so mechanically the great influence for good of rest, or comparative rest, is evident; equally clear is it that where toil is necessitated its effects are injurious. Consequently, the prognosis varies with the affluent and the poor; because an important part of the treatment is impracticable in the case of the latter. Still, rest is no more than one factor, albeit a most important one, in the treatment.

The effects of rest in many cases are so good, so satisfactory, that they produce unfortunate results. Especially is this the case with in-patients in hospitals. A patient with dyspnœa, a cyanozed complexion, feeble, irregular pulse, and passing a small bulk of urine of high specific gravity and saturated with lithates, with or without some œdema of the ankles, is taken in and put to bed by the house surgeon. The treatment is not decided upon, or is deferred till the case is seen by the physician, and in the meantime the patient improves very considerably. His cough is less fatiguing, his respiration less labored, he can lie propped up without great discomfort, perhaps even get a little refreshing sleep, and the pulse is steadier. The improvement is so striking and so unmistakable, that "the expectant treatment" is continued, and in a short time the patient is sufficiently well to leave the hospital and to resume his occupation. Students note the progress of the case, the steady improvement from day to day; and are profoundly impressed with the effects of rest in the relief of disease of the heart. They are apt, too, to form the impression that rest is the

treatment, *pur et simple*, of organic disease of the heart. But let them follow that patient's history a little further, and watch the effects of the resumption of toil, the exertion requisite for the earning of the daily bread; and what do they see? They see every symptom gradually return with its former or even increased force. The heart, which was still equal to the call upon it when the patient is quiet in bed, or even rather more, so that it improved during the period of quiet, is unequal to the demands of toil. The improvement wrought in it by the rest in bed is soon lost under the taxation of labor, and the effects of occupation quickly exhaust the little reserve of force accumulated during the stay in bed. Rest in bed then, valuable though it be, is not the sole treatment to be adopted for those who must work or else they soon must starve; it is of priceless value as an occasional indulgence, and many patients, by means of a few weeks in bed every year, manage to work on with a mutilated or feeble heart for years, without much progress down hill. While fully appraising the value of rest, it is equally necessary to avoid overrating its importance.

Rest is of the greatest value in all forms of organic disease of the heart. Its paramount importance, however, has not been sufficiently recognized in the treatment of acute inflammatory disease in the heart. Acute endocarditis of rheumatic associations is a passing storm, which in itself is of comparatively little moment; but it lights up a growth of connective-tissue corpuscles in the fibrous structures of the valves and chordæ tendineæ immediately beneath it. This goes on after the inflammatory storm has passed over the endocardium, and the acute general symptoms are gone. The patient is convalescing, and, regardless of the lessons of pathology, we forget the cardiac condition and treat the general state; we give the patient tonics, and congratulate ourselves and him on his rapid convalescence; and yet if we had been his bitterest enemy, designing to injure him by malice, we could do nothing more hostile to his true interests. The sooner he is up, the firmer the radial pulse, the greater the strain on the mitral valve every time the ventricle contracts—the same holds good to a less extent of the right side of the heart—the more the strain thrown on the mitral vela and their cords, the more the growth of connective

tissue in them is fostered. It is enough to make one despair of the reasoning powers of the species to see how the lessons of pathology have been forgotten and disregarded by all writers—the writer not excepted in the first edition—in their management of endocarditis. Even in the elaborate work of the late Dr. Sibson, the plan of rest is only followed out during the period of the acute symptoms. Yet physiological rest, or the nearest practical approach to it, is as necessary for an inflamed valve as for a sprained ankle. The patient ought to be kept quiet in bed, and the arterial blood pressure lowered by chloral hydrate, for some time, seven or ten days at least, after the acute symptoms have passed away. By so doing we reduce the strain upon the inflamed valve tissues to the minimum, and so limit the growth of connective-tissue corpuscles, whose subsequent contraction produces such terrible consequences. Such subordination of the immediate to the permanent treatment of rheumatic endocarditis, will save many a patient from a materially injured mitral valve—with all its dire results. Valvulitis is not necessarily progressive, and in many cases a little thickening of the free edge of the mitral flaps is all the morbid consequence left behind by an attack of rheumatic endocarditis.

To arrest and bring to a standstill the parenchymatous inflammation of the valve structures lighted up by acute endocarditis, is what we must directly aim at. To limit the morbid growth at the outset is far more to the patient's interests than to passively permit—if not to actually encourage—a valve lesion to be developed; and then to busy ourselves with aiding the natural attempts at muscular compensation. It is much better to prevent than to imperfectly compensate the mischief wrought by valvulitis. If merely a little thickening or roughening of the free edges of the valves remain in a static, non-progressive condition, then it is easy for the system to develop a sufficient muscular compensation, and the patient is very little the worse. He may have a murmur, but its significance is small; his prospects of life are little deteriorated thereby. In other cases the valve lesion goes on steadily, and even sometimes swiftly, from bad to worse, the stenosis increases, or the regurgitation becomes greater—new muscular compensation is demanded; this fails at last, and the patient

dies with all the sequelæ, painful and dreadful, of a confirmed valvular lesion. In aortic as well as mitral endocarditis, every effort must be made to limit the valvulitis at the time of its manifestation. We have every reason to know that both forms may be arrested and brought to a static condition, under favorable conditions of tissues and of treatment; and this we should aim at by every means in our power. The more so that our measures for increasing the muscular compensation, when valvular disease is once established, are liable to the imputation of increasing the strain on the diseased valves, and so of fostering the morbid changes.

In all valvular disease the failure of the muscular compensation, set up more or less perfectly, is the thing to be dreaded as the beginning of the end. Muscular failure is, then, what we have to prevent if possible in all confirmed disease; dilatation, either primary or secondary to valvular disease, is the condition of the muscular walls of the heart which we must prevent or remedy by every effort in our power. The considerations given in the chapter on hypertrophy and dilatation, must be borne in mind; we must remember that when the demand upon the heart-walls is sharp, or the nutrition defective, dilatation is set up, without hypertrophic growth enough to limit the dilating process. When, then, dilatation is setting in, the acuteness of the demand must be diminished by the adoption of quietude. At the same time the nutrition of the heart tissues must be encouraged by the administration of good nutritive food, with hæmatics, and especially chalybeates. Not only in acute conditions of valvulitis or dilatation are these two matters to be borne in mind, but throughout the whole course of a chronic process must they be remembered. If a man with a diseased heart follow a laborious employment, he must be counselled to abandon it for a less trying one. However, it must not be forgotten that an occupation which makes severe demands at intervals, is more injurious than one which, though it involves longer hours, is less associated with severe effort. The patient must be so placed that he may live as long as possible.

Then, too, in muscular failure, the ventricle is imperfectly emptied at each systole, so much blood remains unexpelled, the

arteries are imperfectly filled, and tissue nutrition is impaired in the heart as well as elsewhere. The indication then is to administer a drug which will increase the energy of the ventricular contractions and fill the arteries. Such an agent we possess in digitalis. Not only does digitalis increase the energy of the ventricular contractions, but it slows the pulse by lengthening the diastole, and so permits of the heart getting a longer sleep. More sleep, a larger supply of arterial blood of better quality, are the main factors, along with lessened toil, by which the dilating process may be arrested; and a restoration of shape or the development of hypertrophic growth be achieved. Imperfect blood nutrition, to whatever due, whether want of food or defective digestion, leads to loss of propulsive power in the heart. Flint ("Diseases of the Heart," 1870) says: "The combination of anæmia and enlargement of the heart is to be prevented if possible, and, if it exist, anæmia, if possible, is to be removed by appropriate measures of medication, diet, and regimen. Irrespective of this condition of the blood, all agencies which tend to weaken unduly the force of the ventricular contractions are contraindicated. In proportion to the weakness of the heart will be the tendency to dilatation rather than to hypertrophy. So long as hypertrophy predominates the patient is comparatively safe" (p. 86). When dilatation is manifesting itself the patient should be confined to bed to reduce to the minimum the demand on the nutritive processes.

This is often necessary after debilitating diseases, or where the heart has been affected, as in certain febrile and inflammatory conditions where myocarditis is found (and where the muscular fibre is too weakened to contract efficiently upon the contents of the ventricular chambers and so stretches), and dilatation is set up. Pretty much the same effects upon the muscular fibre are produced by nervous exhaustion of the sympathetic ganglia by overwork, want of sleep, and similar causes of exhaustion. There is not merely mental tire with sensations of muscular fatigue; the cardiac ganglia are wearied, they explode unrhythmically and with diminished energy; the muscular fibrillæ, also exhausted, respond by imperfect contractions, and then dilatation follows—

which may only be of a temporary character and be readily recovered from.

All conditions which tend to weaken the system must be removed. Indigestion, or mal-assimilation of food, as preventing proper blood formation and nutrition; all debilitating discharges, as diarrhœa (except when the later stages of dropsy is reached), dysentery, hæmorrhoids (except in later stages), must be remedied if possible; and in women menorrhagic and leucorrhœal drains arrested. All exciting mental pursuits must be abandoned or diminished. Quain, B. W. Richardson and others think, not unwisely, that the increased rate at which we live in this "madly striving age" exercises a decided influence in the production of disease of the heart. The increase in the death returns from heart disease is not wholly accounted for by a better recognition of heart disease and a more correct nomenclature; but is due to some extent to the actual increase in the frequency of heart disease. Excitement of all kinds, parties, balls, debauches, and especially sexual excitement in all forms, concerts, theatre-going, too much music in any form, attendance on meetings or services, tobacco-smoking, indulgence in tea, etc., are to be avoided. Frequently will some occult cause of cardiac disturbance or debility be found, which explains the failure of the ordinary treatment in certain cases; when found and attended to the case progresses.

A great number of heart cases presenting themselves to the practitioner, whether of simple muscular failure, or valvular disease, will be found to possess a fair amount of capacity for exertion, or, in other words, are in the early stages of disease. Here, usually, it is only necessary to economize the body-forces, by diminished exertion, the avoidance of all causes of exhaustion, and to resort to some of the combinations of remedies mentioned in the next section, to cure absolutely in a few fortunate cases; and to give great relief in the great majority. Indeed, in some cases the relief furnished by treatment leads to recklessness on the part of the patient.

Two such cases I remember well. One an old Irishwoman in Leeds, who suffered from dropsy and dyspnœa, and who was so quickly and effectually relieved by treatment, that she became quite careless. Relief was immediately followed by the resump-

tion of her pipe and the "cratur." Again and again was she relieved by treatment; but at last careless delay led to great aggravation of her condition, and she was dying when seen. Another case was that of an ex-soldier at the West London Hospital; he had been invalided out of one regiment for heart disease following rheumatic fever. After working for some time he enlisted in another regiment, and served his time out in it. When he came under my care he had mitral regurgitation with an irregular pulse and œdema in the lower limbs. During the course of three years he presented himself some six times, each time seriously ill. Digitalis acted like magic upon him, and the relief given was swift and effectual, and he was back to work in a very little time. All advice was thrown away upon him, and, as I have not seen anything of him for a year, the great probability is he is dead.

But, unfortunately, the tendency of a large proportion of cases is to progress steadily downwards, with greater or less speed; treatment being more or less effectual. A course of digitalis and iron is requisite for some cases only from time to time, with intervals when the patient is so well as to need no treatment. Other cases, again, require digitalis continuously. An old patient of mine in Westmoreland, a woman with Bright's disease, has taken digitalis continuously for twelve years; if she intermits it for a week she is compelled to resume it. During all this period there has been nothing of the character of accumulation. Indeed, nothing has ever occurred in my experience to suggest the idea of an accumulative action in digitalis; and in only two cases have I found the drug to decidedly disagree with the patient, and necessitate the substitution of belladonna in its stead.

The steady and persistent use of digitalis is very beneficial in cases of dilatation of the heart, with or without mitral disease. The natural efforts to establish compensatory hypertrophy are greatly aided by the administration of digitalis combined with chalybeates; and often by the addition of strychnia, which acts both upon the cardiac and the respiratory centres. Such action on the respiratory centres improves respiration, and so acts favorably on the heart. "Reciprocal relations subsist between the vasomotor, cardiac and respiratory centres. Hence, oscillations of the pulse and blood-pressure accompany the respiratory move-

ments, quite independently of any mechanical effect of the movements of the chest on the heart; the pulse being quickened during inspiration and slowed during expiration" (Ferrier).

When heart adynamy is combined with embarrassment of the respiration, as in chronic bronchitis, with or without emphysema, or cirrhosis of the lungs, then the addition of strychnia and carbonate of ammonia (both distinct stimulants to the respiratory centres) to digitalis is very useful in practice.

The history of digitalis is most interesting and instructive. Its use for the relief of dropsy extends backwards till it is lost in antiquity—probably its first use was an accident. According to Pereira foxglove is mentioned in a manuscript written before the Norman Conquest. Fuchsius, in 1542, first gave it the name of digitalis, from "Fingerhut" (the German name), or finger-stall, on account of the shape of its blossom.

Dr. Withering, of Birmingham, in 1785, wrote an excellent monograph on digitalis, from whence dates our proper knowledge of this agent. He observed that in dropsy "it seldom succeeds in men of great natural strength, of tense fibre, of warm skin, of florid complexion, or in those with a tight cordy pulse. On the contrary, if the pulse be feeble or intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcous limbs readily pitting under the pressure of the finger, we may expect the diuretic effects to follow in a kindly manner." It produces its diuretic effects by raising the blood-pressure in the arteries generally, and so increasing the pressure on the thin-walled glomeruli of the kidney, and thus the bulk of urine is increased. Dr. Holland wrote, "the enlarged and flaccid heart, though on first view it might seem the least favorable for the use of the medicine, is, perhaps, not so." At that time the prevalent view of the action of digitalis was that it was a cardiac sedative; that is, it was believed to lower the activity of the heart. From the relief afforded by it in palpitation it acquired the name of "the opium of the heart." To give the reasons why this view is unsound is, now, flogging a dead horse. More correct views now generally obtain; though in sundry books of modern date, digitalis is still spoken of as if it lowered the activity of the ventricular contractions.

The literature on digitalis is enormous.

The names of Boerhaave, Withering, Cullen, the Darwins, Ferrier, Beddoes, Sanders, Orfila, Cruveilhier, Bouillaud, Traube, Homolle and Quevenne, Christison, Pereira, Dybkowsky and Pellikan, Fuller, Handfield Jones, Hilton Fagge and Stevenson, Winogradoff, Lauder Brunton, Balthazar Foster, G. Balfour, H. C. Wood, S. Ringer, Niemeyer, Rosenstein, Ackermann, and others, are associated with investigations into the *modus operandi* of digitalis, and its action upon the heart and vascular system. Other agents possess an allied action. Thus Lauder Brunton has found casca, an ordeal poison of Africa, to have a nearly similar action. Professor Fraser has used the *strophantus hispidus;* P. M. Braidwood found the dajasck, or arrow poison of Borneo, to kill with the heart firmly contracted in systole. The *scilla maritima* also acts powerfully upon the heart, a fact testified to by many observers; its diuretic action, with a feeble pulse, being a well-known clinical fact.

Scoparium, or broom, probably acts in a like manner, and is a drug whose action is well worth investigation. Caffeine has some value; while the action of belladonna is generally admitted. Dr. H. Barnes of Carlisle, and Dr. J. Wallace, now of Liverpool, some years ago made some observations on the action of members of the scrophulariaceæ, but the results have not been published. Dr. Carnecs, in Vienna, many years ago thought the *digitalis lutea* more potent than the *digitalis purpurea* in ordinary use.

Certain investigations have been made by the writer as to the action of digitalis upon the heart. A series of observations were made upon the frog in 1869, and further investigations were made on the guinea-pig and the rabbit in 1875–6, in connection with grants from the funds of the British Medical Association.

The following is an extract from an essay which gained the Fothergillian Gold Medal of the Medical Society of London for 1878, with but some slight verbal alterations. A few words on the action of belladonna and aconite upon the heart are added, as helping to illustrate the matter.

Digitalis.

Some experiments illustrating the action of digitalis upon the heart were performed by me in 1870, and formed the counterpart to the clinical observations on which were founded the conclusions arrived at in my essay on digitalis. The experiments were only of an elementary character, and performed on birds, fishes, and frogs. No calculation was made of the amount of the drug administered in each experiment. The part of the experiments interesting at present is that where I elicited the antagonistic action of digitalis and aconite. Having compared the actions of these two drugs, and finding that digitalis brought the heart to a standstill in firm contraction, while aconite arrested it in full dilatation; the idea of testing their antagonistic action suggested itself. So aconite was given to frogs with their hearts contracted by digitalis; and in others digitalis where the heart was paralyzed by aconite. The aconite did not exercise a very marked effect upon the hearts contracted by digitalis, but the other experiment was very successful. "When all action had apparently ceased, the first effect of the administration of digitalis was to produce an imperfect contraction at long intervals; then the intervals became shorter, and the contractions more complete, some irregularity, both as to time and amount of contraction, being observed. Slowly and gradually, however, the distended ventricle recovered itself under the action of digitalis, the contractions being more rhythmical and perfect, and the distension less and less pronounced, until a return to normal contraction and distension was brought about. If the administration of digitalis were then continued, the same appearances were brought about as when no aconite has been previously given." Attention was drawn to these experiments by the fact that shortly after this Dr. Dobie, of Keighley, administered digitalis to a man dying of aconite poisoning, with the result that the man recovered. To this case reference will be made in a later section of the essay. This line of experiment was followed out by Boehm, who found that muscaria, delphinia, and aconitia would restore cardiac action in the frog when arrested by digitalis poisoning. More recently further experiments as to the

antagonism of digitalis and aconite in warm-blooded animals have been made. They were practical failures, however, as, though the digitalis maintained the action of the heart, it did not prevent the death of the animals from paralysis of the respiration induced by the aconite.

The effects of the experiments of myself and of Boehm went to corroborate the views of Traube and others, that digitalis was not a cardiac sedative in the ordinary sense of that word. That is, it did not calm the heart's action by depressing the activity of its nervous centres. The old view was that digitalis lowered or moderated the heart's action, and it was called "the opium of the heart." But this view has given way to another, viz., that digitalis excites more perfect contraction of the cardiac ventricles. Traube had observed that the effect of digitalis was to increase the blood-pressure in the arteries. Then Malan and myself observed that it contracted the arterioles, while Fuller and Handfield Jones had experimentally ascertained that in animals poisoned by digitalis the heart was firmly contracted, and so contrasted with the state produced by aconite and chloroform. Eulenburg and Ehrenhaus had noted that a frog's heart, when removed from the body and its apex dipped in a solution of digitalis, was so brought to a standstill in systole.

Consequently, the position which digitalis now holds is that of a cardiac tonic, *i. e.*, it increases the energy of the ventricular contractions. It tends, as Niemeyer puts it, to fill the arteries by emptying the veins. Consequently, digitalis is now administered in cases of cardiac failure, where the heart is inadequate to the duty of propelling the blood forward in sufficient quantity. It does not matter what the state of the heart is so much, whether hypertrophied or dilated; the real question is this: Is the heart equal to its work? Even with considerable hypertrophy the heart may still be unequal to its functional duty, and the hypertrophy may be insufficient. Under these circumstances the administration of digitalis is indicated, but in lesser doses than in dilatation. The dose must be proportioned to the natural efforts at compensation. If a fairly good compensatory hypertrophy is being naturally instituted, then small doses of the drug are alone required; if the ventricular chamber is prominently dilating, then

large doses are absolutely necessary. Under the latter circumstances doses of digitalis are well borne and do great good, which would produce very serious symptoms if given where the heart is considerably hypertrophied. The theory propounded by me is this: Digitalis excites more perfect ventricular contraction, the arteries are better filled with blood, and consequently the heart itself is better nourished, while the brief diastolic sleep is lengthened. Betwixt the longer rest and the improved nutrition the heart is enabled to grow and to increase in power. This view is borne out by the observations of other authorities. Traube's observations as to the increased blood-pressure in the arteries produced by digitalis have been corroborated by Lauder Brunton and others, and it is no longer in dispute that the arterial tension is increased by this drug, except in distinct poisoning, when it falls. Thus Balthazar Foster has shown that the slowing of the pulse by digitalis is achieved by lengthening the diastole, not by any effect upon the length of time of the systole. Thus the above view of an improved nutrition and a longer rest is seen to be sound. As long as the blood-pressure in the arteries can be maintained, all is well. The aortic recoil fills the coronary vessels, and the heart is sufficiently supplied with blood, and so its tissue integrity is maintained. But when the blood supply to the heart is unequal to its proper tissue repair, then molecular decay is inaugurated. Mauriac has pointed out how soon the massive hypertrophy of aortic regurgitation fails early, from the blood supply to the coronary vessels becoming impaired from the insufficiency of the aortic valves. Normally these valves arrest the backward flow of the blood on the aortic recoil, and the coronary vessels in the sinuses of Valsalva are well filled with blood; but as the valves become more and more incompetent, the flow into the coronary vessels is diminished, and the heart's nutrition is impaired. Consequently, the prognosis of a case of aortic regurgitation is widely different from that in aortic stenosis, or in mitral disease; because in these latter cases the blood supply to the heart can be pretty well maintained, while in aortic insufficiency it soon fails. These conclusions are confirmed by the clinical observations of Balthazar Foster, that the subsequent history of a case of traumatic rupture of the aortic valves is profoundly modified

by the fact which valve is ruptured. If it be that cusp behind which there is no coronary artery, the case will last comparatively a long time. But if the torn cusp have an artery behind it, then the nutrition of the heart is soon affected, and the case runs its course in much less time.

The use of digitalis in practice is, as nearly as is yet arrived at, as follows: My essay on digitalis* is now out of print, and is not likely to be republished, and the present offers an opportunity of making the views there put forward available for those who might desire to consult the original essay, and are no longer able to do so. When the heart, from any cause, ceases to propel the blood forward into the arteries, the system suffers from the want of arterial blood. The arteries are insufficiently filled, while the veins are surcharged with blood, and a whole series of pathological sequences follow from the venous fulness; among the rest a development of connective tissue in the viscera, as the lungs, the brain, the liver, spleen, and kidneys; there is fulness of the venous radicles of the stomach, and therewith impaired digestion and a constant feeling of "fulness," as if the stomach were distended with a liberal meal, when in reality it is empty; there is albuminuria from congestion of the renal veins, and not rarely serous effusions from the intestinal venules, forming diarrhœa. The organs connected with the valveless portal circulation especially feel any venous congestion from failure in the central organ of the circulation. The tissues, including the muscular structure of the heart itself, are suffering from want of a sufficiency of arterial blood charged with nutritive material. When digitalis is administered, the ventricular contractions become more complete and the arteries are better filled and consequently the veins more thoroughly emptied. When there is pronounced dilatation of the heart, and a little blood is pumped out only at each systole, and the heart is soon full to distension, it quickly contracts with an inefficient systole, the pulse being rapid, feeble, and compressible. Thus it is that digitalis so promply affects the pulse-rate. By its effects upon the cardiac ganglia it causes a slower

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* Digitalis: its Mode of Action and its Use, to which was awarded the Hastings Gold Medal of the British Medical Association for 1870.

but more energetic discharge of force, and with it a more powerful muscular contraction. The systole is more complete, the chamber is more efficiently emptied, and consequently is not so soon refilled; so that the requirements of the ventricle in diastole correspond to the slower rhythmic discharges, and a slower pulse-rate is established; while the pulse is firmer and less compressible, the arteries are better filled with blood. At times digitalis will notably lower the pulse-rate under other circumstances than those just mentioned, illustrating its effects upon the discharging cardiac ganglia. When the pulse-rate falls very markedly under its use, as when it falls below 50, it would be well to substitute belladonna, squill, strychnia, or casca for it.

"The series of altered actions consequent upon increased ventricular contraction run in the following order or sequences; and it may be desirable for the sake of lucidity to arrange them in a series of propositions, each depending on the one before, like a logical syllogism. The effects of increased contraction, then, are:

"1. Increased arterial distension and tension, which give relief to the systemic symptoms, and further causes,

"2. Increased arterial recoil. This is the propelling power for the coronary arteries; and thus increased arterial recoil means,

"3. Increased or improved coronary circulation; and this, in its turn, produces,

"4. Increased nutrition of the heart, which results in

"5. Compensatory hypertrophy.

"Not only is improved nutrition of the tissues of the heart thus secured, but the brief diastolic sleep is lengthened; and betwixt a longer sleep and an improved nutrition the heart structure is repaired efficiently, and recovers its tone more or less completely, according to the nature of the case."

Consequently, as said before, in fairly good hypertrophy, digitalis is either not indicated at all, or, if so, only in small doses. As digitalis not only acts powerfully on the heart itself, but also produces contraction of the peripheral arterioles, and so raises the blood pressure in the arteries, it should not be given in hypertrophy in large doses, especially where the arteries are atheromatous, as it greatly increases the risk of apoplexy from rupture of

an encephalic artery. Such an accident has occurred from the use of digitalis, even when the hypertrophy was commencing to fail in incipient fatty degeneration.

In dilatation, however, its use may be free, not only without danger, but with positive advantage. "In dilatation, where the system is confessedly unequal to the establishment of compensatory hypertrophy, the use of a drug whose action is to throw the ventricle into a state of tonic contraction is readily understood as being of the greatest service; and this also explains how its use in large and continued doses is not only not productive of symptoms of poisoning, *i.e.*, of ventricular spasm, which was once imagined to be due to an accumulation of the drug in the system in some mysterious inexplicable manner,*—but is even necessary to the continuance of life. Without it, or some similarly acting drug, dilatation must become only more and more extensive, and hand-in-hand with that extension must be an increasing inability on the part of the heart to recover itself; hence, still more enfeebled circulation, blood stasis and its consequences, until the widening vicious circle ends in somatic death." It may, indeed, be questioned, if any risk can be incurred by the free administration of digitalis in cardiac dilatation until the dilatation is notably reduced. In the case of a girl, M. C., æt. 19, with aortic regurgitation and an enormous dilatation of the left ventricle, whom I sent into St. Mary's Hospital under Dr. Broadbent, the use of digitalis in the hospital was followed by a reduction of the dilated ventricle of a very pronounced character. After which severe palpitation came on, and ceased when the digitalis was withdrawn. On coming out of the hospital and again moving about, it became necessary to resume the digitalis, which she continues in small doses still, now three and a half years since she was in St. Mary's. In the case of a huge man, a patient at Victoria Park Hospital, with an enormously dilated heart, without any valvular mischief, it has become needful to exhibit digitalis in unusually large doses (Tinct. digitalis ♍xx;

* There is no accumulative action connected with digitalis any more than with belladonna, strychnine, arsenic, or mercury. If these agents be taken into the system in doses in excess of the eliminative powers, they "accumulate," and produce toxic symptoms. But they do not "lie in wait" to work mischief.

Tinct. nucis. vom. ℞xv; ter in die) to enable him to work—for rest he will not. He since this is dead. Dropsical symptoms set in before he died. Commonly this reduction of a dilated heart—and with it the size of the mitral ostium—will enable partially injured mitral vela to close the ostium completely on the ventricular systole; that is, these valves are rendered once more functionally competent. At other times the reduction of a widely dilated ventricle, with corresponding augmentation of its power, will develop a mitral systolic murmur previously inaudible. In cardiac dilatation digitalis may be freely exhibited, and if the practitioner feel somewhat afraid of such use of it, he may combine it with strychnine and carbonate of ammonia—an addition which ought to relieve his mind of every apprehension.

In valvular disease of the heart, digitalis may be resorted to with great advantage; but there are points in each form of valvular lesion to be attended to, which are important enough to make mention of them desirable. To take them in order is probably the best plan.

Aortic Obstruction.—Here there is a distinct obstacle to be overcome, namely, a narrowing of the aortic orifice. This is usually achieved by the natural powers alone, and aortic obstruction commonly does not come under the notice of the physician unless the patient has been pulled down by some intercurrent condition or malady. In elderly persons, however, it presents itself primarily with certain objective and subjective phenomena. Here a new balance can only be effectually struck by an increase in the driving power, so that the normal amount of blood may be driven through a narrowed orifice in the normal time. According to the view that digitalis slows or retards the systole, and so permits the blood in the ventricle to be completely passed through the narrowed orifice, the wants of the system would be no more perfectly met than before. But against this view are the facts, that in unaided hypertrophic compensation in aortic stenosis there is no such elongation of the systole, and that Balthazar Foster has shown that digitalis does not slow the systole, but lengthens the diastole. In aortic stenosis, digitalis is indicated in small or medium doses according as the hypertrophied ventricle can or can not quite successfully meet the obstruction

offered. If the ventricle cannot completely empty itself, and blood is accumulating in the left ventricle, *i. e.*, a condition of dilatation is being established, then digitalis is clearly indicated. When there is no dilatation, and the compensation is sufficient, digitalis is not required.

Aortic Regurgitation.—Here a complex problem is presented to us, for the hypertrophy here, often enormous, is to antagonize the increased internal pressure upon the heart-walls during diastole; not to overcome any obstruction offered. There being a decided increase in the distending force, there is dilatation in spite of the massive hypertrophic growth. This enlargement of the left ventricle, in capaciousness as well as in power, causes at each systole a largely increased mass of blood to be suddenly thrown into the arteries on each ventricular systole, and that, too, with abnormal force. Consequently the arteries are overdistended, and become ruined rapidly by atheroma. The hypertrophy which limits dilatation in its turn does harm, and some writers call this condition "overcompensation." Here, and at this stage, digitalis, which would lengthen the diastole and increase the vigor of the ventricular systole, is contraindicated. A drug of opposite action, which would shorten the diastole and diminish the vigor of the systolic contractions, would be desirable. But in the later stages, when the hypertrophy is failing and the heart-walls undergoing decay by fatty degeneration, are once more yielding to the distending force, then digitalis is indicated to prolong life. The danger of sudden death by overdistension of the failing ventricle in diastole is great in the later stages of aortic regurgitation, and here digitalis is useful; but each case requires its own management and not rules of thumb.

Mitral Disease.—In the consideration of the use of digitalis in mitral disease, it will serve no good practical end to separate stenosis and regurgitation. In the one case the blood is obstructed by a narrow orifice, in the other it is dammed up by a backward-flowing current. The results are the same. The auricle is dilated, the flow of blood in the pulmonic circulation impeded, the pulmonary artery and its branches are dilated and thickened; the obstruction in the pulmonic circulation leads to an enlargement of the right ventricle, hypertrophy, with more or less dila-

tation. So long as the right ventricle can contract efficiently upon its contents the blood is driven forward successfully, and the increased obstruction met. When this capacity of the right ventricle is naturally established, all is well; but when the system unaided is unequal to such compensation, or the compensatory hypertrophy is failing, then digitalis is of the greatest service. There is no difference of opinion in the profession about the utility of digitalis in mitral disease. By its effects in producing more perfect ventricular contraction it exercises the most beneficial action upon the failing right ventricle. Where there is accompanying bronchorrhœa digitalis is not contraindicated, but the reverse. The bronchial flow is not the measure of the blood-pressure in the pulmonary circulation, but of the stagnation in the bronchial veins, a part of the general venous congestion; and the administration of digitalis lessens the bronchial flux, while it raises the blood-pressure in the pulmonary vessels.

Right-side Dilatation.—Digitalis is of the greatest service in dilatation and failing power in the chambers of the right heart, whether from mitral disease or chronic changes in the lungs, as cirrhosis of them, or where there is chronic bronchitis or emphysema, or chronic pleuritic effusion, or other cause of obstruction to the blood-flow in the pulmonic circulation. In such cases it is well to bear in mind the coexistent embarrassment of the respiratory centres and to combine with digitalis ammonia, nux vomica, or belladonna.

Tricuspid Regurgitation.—When the tricuspid valve has become insufficient, then our efforts carry with them little success, because we have no muscular chamber behind the lesion to be acted upon, the muscular fibre in the venæ cavæ being but small and powerless. Still it may be tried in combination with brisk purgation to relieve the portal circulation.

In *temporary cardiac conditions* of asthenia digitalis is useful, as in cardiac dyspnœa, with overdistension of the right ventricle. If the overdistension is threatening paralysis in diastole, it may be well to relieve it by venesection, giving ammonia and digitalis at the same time. When the right ventricle has been overstrained by long-continued efforts, as in long runs, diving, etc., digitalis is useful. The right ventricle is apt to become overstrained

where the breath is long held or the circulation in the lungs impeded; and the person is less equal to an effort, instead of more equal; this, in sporting phrase, is termed to "train off," in contradistinction to continued improvement and capacity, which is termed "training on." It is worth while trying the effects of digitalis in horses whose wind has been impaired by long severe effort (overdistension of the right heart), or even in broken-windedness from emphysema. Of course in the latter case only some improvement can be looked for, but in overdistension of the right heart even more seems potentially attainable. Of course stimulants to the respiratory centres at the same time are indicated.

In *Shock*, digitalis with ammonia and nux vomica is likely to be of much service, and it has already been so used with good effects.

Palpitation.—In palpitation due to exhaustion, or to muscular failure, digitalis is very useful. Here palpitation is the active expression of incapacity in the heart. It is overtaxation, not overaction, with which palpitation is here associated. It may occur with a heart dilated, hypertrophied, or normal, but it indicates want of power. Here digitalis and iron, with rest, are clearly indicated. In the palpitation of gout, and in those forms wherever it is truly neurosal, or of reflex origin, digitalis does little good.

Fatty Degeneration of the Heart.—Here digitalis can only be of use for its action (through the cardiac ganglia) upon the muscular fibres remaining sound. Where the heart is failing obviously and losing its rhythm, digitalis will be of service. There are two points to be considered here, though, and they are both related to the question of internal pressure. Firstly, if the degeneration be unequal throughout the heart, to increase the blood-pressure in systole is to increase the pressure on weakened spots, and so to cause cardiac aneurism, or even rupture,—or at least to run the risk of doing so. Next, fatty degeneration of the heart is usually associated with advanced atheroma of the vessels, and so is "a preservative lesion," and where so associated, to increase the vigor of the heart's contractions on the one hand and tighten the arterioles on the other, would lead to an increase of the in-

ternal pressure within the arteries, and so to markedly increase the danger of rupturing them. Still here it is usually desirable to administer digitalis, in spite of these theoretical objections.

In cases of anæmia with unfilled vessels it will usually be found advantageous to give digitalis. By increasing the activity of the heart and contracting the arterioles it tightens the vascular walls upon their fluid contents, and so raises the blood-pressure in the arteries. Consequently it is of great service in cerebral anæmia, along with tonics and hæmatics.

In considering the action of digitalis it must be borne in mind that it not only acts upon the cardiac ganglia, and so excites more perfect contraction of the heart itself, but that it acts also on the vasomotor centre, and produces contraction of the peripheral arterioles. If it did not possess this second action digitalis would be of little therapeutic use, for as fast as the ventricle threw blood into the arteries it would escape by the open arterioles. By obstructing the outflow, as well as increasing the blood-flow into the arteries, digitalis raises the blood-pressure in the arteries. It fills the arteries, and in doing so empties the veins. It thus relieves the symptoms consequent upon arterial anæmia. The flow of urine is ordinarily the measure of the arterial fulness. As Traube pointed out, the bulk of urine is the measure of the blood-pressure on the renal glomeruli, and this usually is in direct proportions to the general blood-pressure. Consequently many persons who have not carefully studied the mode of action of digitalis say: "It always does good when the bulk of urine is increased;" or: "It always does good when its diuretic action is established." Quite true! But this diuretic action is really the evidence of its effect upon the circulation. When it increases the bulk of urine digitalis is producing its desired effect upon the circulation.

There is just one speculative point to be noted in the action of digitalis, and it is this: If digitalis contracts the terminal arterioles, it must cause further obstruction to an already enfeebled heart, it may be argued. This is quite true. But when the heart is distended its sensory nerve throws the vaso-inhibitory nerves into action, and so dilates the peripheral arterioles. Digitalis restores them to their normal calibre, while it acts directly

upon the heart, increasing the vigor of its contractions. Any risk of increasing the obstruction offered to the ventricular systole, so as to embarrass the heart, must be regarded as hypothetical rather than practical; like the dangers of its use in fatty degeneration.

From its effect upon the heart and arterioles, digitalis is used to lower the body-temperature, in certain pyretic conditions where the arterial tension is low. According to Ackermann and Heidenhain the temperature and the blood-pressure vary inversely; when the blood-pressure is raised, the temperature falls, when the arterial tension falls, the temperature rises. Digitalis has been found very useful in pyretic states where the heart is likely to fail, as in typhoid pneumonia, relapsing fever, etc. The modern use of digitalis has been reached as much by the aid of experiment as clinical observation. Indeed clinical observation, as the history of the drug shows, absolutely needed the aid of experimentation to direct it aright.

The history of digitalis, as a therapeutic agent, is most instructive. The views taken of its action represent the pathological opinions of the time. When hypertrophy was a wanton and useless, indeed dangerous overgrowth, digitalis was a sedative, because it quieted the tumultuous action of the heart. Now, when we recognize that hypertrophy is a useful compensatory growth, we discover that digitalis is a stimulant to cardiac action, and quiets the heart by enabling it to contract more efficiently, and therefore more steadily. The whole question of acting upon the heart in the way of increasing its power is quite a recent one. It is now acknowledged generally that we can stimulate the heart to contract more efficiently by means of digitalis, belladonna, and strychnia. It will be seen that not only can we do this, but that there are excellent grounds for holding the view that now we are in a position to systematically and designedly excite more perfect respiratory action when it is desirable to do so.

We possess agents which will stimulate the nervous centres of respiration when becoming embarrassed, so that they will once more respond to the natural stimuli to action. It will be seen that the agents which possess this power are also those which act

upon the circulation, and some difficulty will arise from this combined action; nevertheless it will be possible to demonstrate that there exist drugs by which the respiratory centres can be stimulated and excited into more perfect action when failing. These agents are ammonia, belladonna, and strychnia, which are not expectorants in the sense that they exercise any influence over the bronchial secretion, but that they affect the nervous centres of the respiratory organs and excite more perfect respiratory action. We can maintain the respiration when failing in acute bronchitis as certainly as we can excite cardiac activity in syncope, or when the heart is failing in acute disease. Probably any action upon one system involves action on the other, but that will not constitute an insuperable difficulty in the inquiry.

Digitalis produces its effects upon the heart, not by acting upon the muscular structures, but through the nervous centres which excite muscular action. It is a direct stimulant to these centres, in that it increases the energy of their rhythmic discharges. It causes these discharges to become more energetic, and under certain circumstances much slower. Nerve centres when becoming exhausted usually become irritable, that is, they act rapidly and with little energy. Under these circumstances a direct stimulant to them lowers the number of rhythmic discharges, but increases them in power. Digitalis and belladonna have a very powerful action upon these cardiac centres, and so has strychnia; while aconite depresses them, as also do the well-known neurotic sedatives, opium, chloral, and bromide of potassium. When we wish to stimulate the energy of these cardiac motor centres we give digitalis, belladonna, or strychnia. Thus, in cases of failure of the heart, these agents are indicated and do good. In the palpitation of the muscular failure they are of service; but in the palpitation of nervous excitement, where the cardiac centres are acting too energetically, they are contraindicated, and sedatives should be used. We are so accustomed to think and speak of the heart from its muscular side only, that we are apt to overlook in thought, and certainly omit in speech, the intimate connection existing betwixt the heart and its nervous centres. Yet all these agents act upon the muscular walls through the nerve ganglia which preside over them.

Belladonna.

Belladonna acts powerfully upon the heart, and has long been a favorite agent in conditions of palpitation of a chronic character. Dr. John Harley states that it causes a rise of blood-pressure in the arteries, and that therefore it is the drug *par excellence* to be used as a diuretic agent when the arterial pressure is low; Dr. Graves commended its use in typhus fever in the advanced stages; and many writers have extolled its utility in conditions of circulatory debility. From what we shall shortly see of its action upon the respiratory centre it is an agent undoubtedly called for when the circulation and respiration are failing. It has been resorted to very successfully in the treatment of collapse by Reinhard Weber, M.D. ("Philadelphia Medical Times," February 2d, 1878), and by Dr. Hodgen, of St. Louis ("Practitioner," November, 1878).

Aconite.

The action of aconite upon the heart is a very powerful and very decided one. A moderate toxic dose produces at first a reduction in the number of heart pulsations, then an increase with evident loss of power, and finally irregular systolic movements with very long intervening pauses ending in diastolic arrest (Achscharumou, Böhm, and Wartmann). According to these latter authorities in aconite poisoning the force of each individual beat is lessened; and after death the cardiac muscle fails entirely to respond to galvanic irritation—its contractility is lost.

Aconite has been used of late very largely for the purpose of quieting the circulation when excited, especially in febrile conditions. Sidney Ringer has done much to bring this agent into notice for the alleviation of pyretic states, especially in children. It lowers the action of the heart, and also of the respiration, and so lessens the chemical interchanges going on; at the same time that it dilates the cutaneous vessels and throws the sudoriparous glands into action, and so increases heat loss. Its effects are very distinct, and for children it really is an excellent measure. In

my experiments I found the younger rabbits to require a larger quantity of aconite to the pound weight to produce a lethal effect than in older rabbits. (One-three-hundredth ($\frac{1}{300}$) of a grain of aconite was a lethal dose for a 3lb. rabbit; while for a rabbit weighing $1\frac{1}{2}$lbs. not a six-hundredth ($\frac{1}{600}$), but a four-hundredth ($\frac{1}{400}$) of a grain was a lethal dose.) The same holds good in man, and Dr. Farquharson recommends great caution in its use in the aged, and extols its utility in children. Aconite, then, can be usefully employed when we desire to lower the heart's action when excited. It affects the muscular contractions through the nervous ganglia, whose discharging intensity it lessens.

These three drugs just enumerated exercise an influence over the circulation and respiration which is very marked and out of all proportion to their action upon other parts of the nervous system. Other agents, as strychnia, as an excitant, and opium, chloral, bromide of potassium, as sedatives, exercise the same action upon the cardiac centres that they do upon the nervous system generally, and may be resorted to with advantage in many abnormal conditions.

Such, then, are our means of acting directly upon the heart. Some formulæ may be appended:

Tinct. digitalis, ♍x.
Sp. chloroform, ♍xx.
Inf. buchu, ℥j.

is a very pleasant mixture, suitable to a weak heart in elderly persons. A more potent mixture is

Am. carb., gr. v.
Tinct. nuc. vom., ♍x.
Tinct. digitalis, ♍xv.
Inf. buchu, ℥j.

Where there are lithates in abundance

Pot. bicarb, gr. x.
vel Sodæ pot. tart., ʒj.
Tinct. nuc. vom., ♍x.
Tinct. digital., ♍x.
Aq. menth. pip., ℥j.

is a good combination. Or this mixture may be given with a gouty taint:

Tinct. digital., ♏x.
Sp. æth. nit., ʒj.;
Tinct. sem. colchici, ♏x.
Inf. buchu, ℥j.

Each form to be taken three times a day.

Where digitalis must be taken continuously for months, or even years, it is well to give it in pill, as by this means the nauseous taste is avoided, while the litter of empty medicine bottles is escaped thereby.

Pulv. digitalis, ʒj.
Fer. sulph. exsic., ℈ij.
Strychniæ, gr. ij.
Pulv. pip. nig., ʒij.
Ext. gentian.
vel Pil. aloe et myrrh, ʒij. M. fl.
Mas.: in pil. 60 div.; i, ibis in die.

Each pill to be taken after a meal.

Digitalis is the therapeutic agent to use in organic disease of the heart; while belladonna seems rather suited to the nervous disturbances of the heart. In some cases bromide of potassium may be combined with either agent.

It is not always easy amidst a congeries of symptoms to say how much of the heart disturbance is due to the organic disease present, how much to nervous disturbance, direct or reflex. And yet it is well to separate the two if possible, and allow for each in prescribing. Digitalis will do a feeble heart good; while bromide of potassium will do much to reduce the effects of perturbation arising from ovarian, or uterine, or other far away irritation. Consequently in some cases the combination is advantageous. It is well to bear in mind the effects of toil upon the heart. When the patient leaves a hospital and commences to work, the dose of digitalis will have to be increased, in order to compensate the effects of toil.

Digitalis occasionally upsets the stomach; it is more apt to do

this when given before meals than after. Where there is gastro-intestinal disturbance it may be given with bismuth and alkalies.

Eulenberg has found the applied electric current useful in giving tone to the heart. The effect of a feeble electric current is to increase the force and lessen the frequency of the heart's action; as the writer has observed with a small Stohrer's battery, one pole placed over the heart, the other to the nape of the neck. This then may be used along with a course of digitalis and chalybeates.

Such are the means of acting directly upon the heart itself by increasing the energy of its contractions. But in addition to these measures, which may be continued persistently for years, there are the means to be adopted when the heart is temporarily embarrassed, as in cardiac dyspnœa. The diseased heart is liable to attacks of acute embarrassment. The right ventricle is overdistended and fails to propel the blood through the lungs, there is pulmonary engorgement; little blood is passed over to the left heart, hence the pulse is small; the irregularity in the pulse is probably communicated from the right heart to the left, rather than originating in the fibres of the left ventricle itself; the veins are turgid and pulsate from the contractions of the distended right heart. Such attacks commonly come on at night. The starting-point of the attack it is not always easy to determine. They are often excited by unsuitable food at supper, and by flatulence. There is orthopnœa, and the patient labors for breath with all the supplementary muscles of respiration in violent action. The features are cyanosed and the lips blue. The danger here is failure of the right ventricle. (How far there is also failure of the diaphragm is yet unknown. Callender has given an account of some cases of fatty degeneration of the diaphragm, in the "Lancet" of January 12, 1867, but in no instance was the disease associated with heart disease.) The line of treatment to be adopted is to act at once on the heart by the application of heat. Large linseed-meal poultices should be applied to the chest, back and front; a layer of double flannel being placed betwixt the poultice and the skin to prevent scalding. The alcoholic stimulants indicated in such a condition should be given with hot water; as the heat is readily communicated from the stomach to the right heart. Rapidly diffusible stimulants, as carbonate of ammonia and spirits of chlo-

roform, with digitalis, should be given freely; and now I should feel inclined to add some tincture of nux vomica. The alcohol and ammonia should be given in doses proportioned to the patient's danger; for the danger is imminent, and for the time the attack lasts, it is often "neck or nothing." When the attack is over, the patient is exhausted and falls back prostrated; not uncommonly sleep follows. These attacks are associated with failure of the right heart. The sense of shortness of breath and discomfort, disagreeable enough to a person in health, when the stomach is distended with gas, or other contents, is intensified in a person with a diseased right ventricle. The descent of the diaphragm is impeded, and the heart is pressed upon by the elastic contents of the distended stomach. (The same thing takes place from accumulation of gas or fluids in the transverse colon.) The space in which the heart must beat is infringed upon by the diaphragm being pushed upwards, and so the heart, and especially the right heart, is embarrassed. The patient may get up into a chair, thus allowing the contents of the abdomen to fall away from the diaphragm, and so attain relief. In some cases of advanced heart disease, the patient abandons the recumbent posture altogether, and lives for weeks in an arm-chair. The arms of the chair are points on which the elbows can rest, and so the shoulders be fixed, and thus the pectoral and other accessory respiratory muscles be more effectually utilized for respiratory purposes. A favorite position with others is to sit with the arms resting on the back of a chair in front so as to fix the chest. When the attack comes on in bed, the patient may get on to the hands and knees, and uncomfortable as the position is, retain it for hours. This position both fixes the shoulders and allows the diaphragm to move more freely. All pressure from without impedes the heart's action, and by its removal the heart is relieved. At times there is an abundant production of gas in the stomach, which comes up in loud eructations; and this distressing condition may last for hours.

Then comes the question of bloodletting. Venesection may be used to relieve high arterial tension, with a bounding incompressible pulse: this is commonly found with an hypertrophied left ventricle. And bleeding may be indicated to protect the arteries,

especially the encephalic, from rupture. But bleeding may also be resorted to to relieve an overdistended right heart. In cardiac asthma the right ventricle is on the point of paralysis from over-distension, and cannot efficiently contract upon its contents; the withdrawal of a little blood from a vein lessens the pressure within the right heart, and it once more can contract upon its contents, and so recovers itself. I remember well, many years ago, when a student, seeing my late father bleed a man in an attack of acute cardiac asthma, the bronchial secretion which was expectorated being tinged pink with blood, from fulness of the bronchial veins. As soon as the blood began to flow the expectoration ceased, and the man experienced relief. The case was one of progressive mitral regurgitation from rheumatic fever late in life. In another case, the writer has several times stood, lancet in hand, watching the effects of stimulants and digitalis, ready to open a vein if relief were not speedily afforded by the other measures, and returned the lancet to its case unused, the attack passing away. The application of hot poultices over a large area has the effect of dilating the cutaneous capillaries, and so of "bleeding the patient into his own vessels," without abstracting the blood from the system, where it is sorely needed. This is better than venesection; but the latter may be imperatively called for, and bleeding will come back into practice under certain well-defined sets of circumstances. It is proper, under these circumstances, to combine bleeding with the administration of stimulants. "While the heart is flapping and fluttering, and its feeble movements can scarcely be felt, and the blood is almost stagnant in the vessels going to it and from it, and almost stagnant in the lungs, we cup and we leech, and we may even venture to use the lancet, and let out two or three ounces of blood from a large vein; but in the meantime we must give our patient wine or brandy" (Latham).

But attacks of cardiac disturbance of an acute character are not always due to primary failure in the heart itself. As we saw in the last chapter, vasomotor disturbance may affect the heart secondarily, producing palpitation and even breast-pang, and dilatation of the peripheral arterioles is the most efficient means of relieving the heart from its embarrassment. Other causes may

disturb the heart. Thus Stokes writes: "Gouty palpitation, hysterical or nervous affections, cardiac attacks, depending on sympathy with the stomach or liver, present the most striking examples, not only of irregularity but of excited action, and these cases may occur independent of any valvular disease, or, if such exist, it is inconstant in its seat, nature, and amount, and incompetent to explain the condition in question. We too often find physicians giving an erroneous opinion from ignorance of these facts, for in their minds the ideas of irregular action and of valvular disease are so closely combined that they make the diagnosis of incurable disorder in cases where an emetic, an antinervous draught, the occasion of gout at the extremities, or a few doses of a mercurial will restore the natural action of the heart." He then relates the case of a lady, who for long was subject to severe attacks of palpitation, accompanied by a loud bellows murmur. After an attack which lasted some weeks, Dr. Stokes saw her again; "the heart's action was perfectly tranquil, the pulse natural, and every trace of murmur had disappeared. Several years afterwards I saw the lady; she was then in perfect health, and mentioned with a good deal of complaisance, that she had not only puzzled her physicians, but that she had discovered her own cure, and this was in the use of an emetic at the commencement of each attack; a practice to which she had been led by the occurrence of accidental vomiting from the effect of some medicine which had been administered." The effect of emetics was to reduce the paroxysms, which became less and less, till at last they disappeared.

In some cases of palpitation, both in organic disease and in mere functional disturbance, the administration of morphia, especially by hypodermic injection, has been practiced, not without success.

Heberden, and others after him, gave opiates freely in angina pectoris, and combined with diffusible stimulants, opium gives much relief in certain forms of cardiac disturbance. But the cases for its use must be carefully discriminated. There is no evidence that morphia aids the heart to contract; indeed the effect of opium and its derivatives is to paralyze the heart, but we have reasons to suppose that it relaxes spasm. In cases, then, where there is

arteriole spasm causing impeded blood-flow, and palpitation is the evidence of the heart's struggles to force on the blood, then morphia will be of service. It is more liable to be useful where the arteriole obstruction is hysterical (Eichwald) than where it is due to lithiasis. In the latter case, to wash out the nitrogenized waste by alkalies in buchu is probably the best plan of treatment; for opium, like mercury, is often badly borne by those who have chronic kidney disease. In one case where morphia was badly borne, B. W. Richardson placed a mustard blister on the neck, "so as indirectly to influence the sympathetics," and the relief afforded was immediate. The principle of treatment in such forms of cardiac disturbance is to dilate the peripheral arterioles, and thus to permit of the ready outflow of blood from the arteries. Inhalations of amyl nitrate do this most effectually and are free from danger.

But when the heart is seriously embarrassed in true cardiac dyspnœa, and the right ventricle is nearly paralyzed by overdistension, or when the powers are failing, and the patient is utterly worn out from want of sleep—sleepy to a degree, dozing off for a moment and then waking up with a start in terror, feeling asphyxiated, and gasping for breath; then opiates must be avoided. The patient could sleep well enough if the necessity for voluntary efforts at respiration did not debar him. Here to sleep is to die. If the patient fall asleep, it is the sleep which knows no wakening. The late Hyde Salter pointed this out strongly. "Take away," he says, "the necessity for the voluntary effort indispensable to respiration, and the patient would soon be so sound asleep it would be difficult to wake him. Suspend the voluntary efforts by an opiate, and the sleep is the sleep of death." However much we may pity the sufferer, we cannot feel justified in prescribing opium, or other narcotic, or sedative, when the severe voluntary efforts are essential to the maintenance of existence.

Chloral hydrate, which is allied to opium in its effects upon the circulation and the respiration, is to be avoided even more studiously than opium, where the heart is failing and its permanent arrest in diastole imminent. Even under less serious circumstances, its use is too often pernicious. A patient admitted into the West London Hospital with aortic stenosis, and consid-

erable enlargement of the left ventricle, was put upon digitalis and ammonia. Yet under this plan of treatment, and rest in bed, he became subject to attacks of severe cardiac dyspnœa; from which he had not previously suffered. On searching for an explanation of this strange fact I found an unreflecting house-surgeon had benevolently ordered him some chloral at bedtime, as he complained of sleeplessness. On stopping the chloral draught the attacks of dyspnœa at once ceased, and never returned, though the patient sank. On the post-mortem examination a tight aortic stenosis was found, with a considerably hypertrophied left ventricle, of healthy texture. The effect of the chloral upon the cardiac ganglia was to paralyze (partially) this powerful ventricle. Where there is no stenosis at the aortic orifice chloral may be less injurious; if it depresses the heart's action it at the same time dilates the peripheral arterioles; but in aortic stenosis it lowers the action of the heart, while it can exercise no influence over the structures of the narrowed ostium.

In the treatment of organic disease of the heart it is just as necessary to avoid some drugs, as it is imperatively necessary to employ others.

Next come the matters of hæmatics, then that of food, and finally that of alcohol. In chronic heart disease it is of the utmost importance to keep the blood of good quality, for tissue nutrition is an important factor in the treatment. The digestive and assimilative organs must be attended to, and the bowels kept open. "The due regulation of the digestive organs, the promotion of all the natural secretions and excretions, and keeping the mass of blood moderate in quantity, but without impoverishing its quality, are objects never to be lost sight of" ("Library of Medicine").

An occasional purgative, and the removal of some fluid from the bowels, will often aid the action of the diuretics employed, especially in the later stages of each case. Hæmatics are not a synonymous term for chalybeates; hæmatics are agents which improve the quality of the blood—no matter how. The steady administration of iron, along with digitalis and strychnia, is indicated in most forms of heart disease, especially in young subjects. In advanced life vegetable tonics are rather indicated than

iron, and with many old people iron rather does harm than good. With old patients it is well to give the vegetable tonics with alkalies; while in young people they may advantageously be combined with acids and chalybeates. It is well to give iron after food; while, if the loss of appetite require bitters, with or without carminatives and aromatics, they may be given before food. Thus a mixture of quinine or strychnine with phosphoric or other acid may be given in infusion of cascarilla before food, and a pill of iron and digitalis after meals with advantage. It is of comparatively little moment which preparation of iron is used, provided it is assimilated. If one form disagrees, try another. Often the muriate of iron (ten drops) in acetate of ammonia (one drachm) will be digested when other forms have failed. The dialyzed iron probably will agree where any form of iron is tolerated. Arsenic may often be added with advantage to the steel mixture, or pill. With old persons, arsenic is commonly indicated as a tonic; especially where quinine and strychnine irritate the bladder centres too much.

Diuretics are hæmatics in the sense that they get rid of much water from the blood. But they, with diaphoretics and cathartics, will be considered in a later section of this chapter. In order to keep the bowels open, it is well to give the iron and digitalis in pil. aloe. et myrrh; or, if a stronger purgative be indicated, ext. aloes aquos; or equal quantities of these two pill-masses.

As to the food, it must fulfil two requisites,—it must be highly nutritious, and it must be readily assimilable. The digestive organs are enfeebled, and the appetite capricious. It is very desirable, then, that the food should be appetizing in form, so far as is possible. Instead of asking invalids what they will have, or telling them beforehand that they are to have such and such a thing, thus exciting their anticipations, till when the reality comes it falls short of their expectations, it is well to prepare a small quantity of something nice, and take it to them without previously saying anything; and then the delight occasioned by the sight of the appetizing morsel induces the invalid readily to take it. As to quantity, it is better to prepare just so much that the patient finishes it, expressing the opinion that a little more would have been acceptable, than to eat till satiated, and yet leave some

uneaten. The first stimulates the appetite, the latter cloys it. A few simple foods may be mentioned. Beef tea alone is scarcely a food; it should be given with oatmeal, boiled sago, or biscuit powder; the same holds good of Liebig's essence. It is better to thicken the fluid with boiled arrowroot than dissolved isinglass. Milk may be taken alone, or with an admixture of some mineral water, as Apollinaris or seltzer water. Milk puddings, stewed fruit and cream, custards, and creams, are suitable food; sponge cakes, jam rolls, and pastry, are unsuitable food. A little fish nicely cooked, as red mullett or whiting, brill, or turbot pudding, will usually be acceptable. Plain meat, with gravy and well-cooked potatoes, or game, may be permitted in cases where the appetite is good and the digestive powers fair. When the case is in an advanced stage the fulness of the venules extends to the gastric circulation. The patient then complains of ever feeling "too full," even before food is taken. If this venous fulness cannot be removed by the general treatment, then the matter of the dietary becomes very important. Good coffee and cream, hot in winter, and iced in summer, has much to be said for it. Milk may be taken with a little sherry or brandy in it as a beverage; while, in winter, milk made hot with treacle and a little ginger, and even a little spirit of some kind, forms a palatable and nutritive drink. Supper is a very important subject; it should be light—a biscuit and butter, with a glass of malt liquor, or sherry, or milk, or beef tea, if preferred, in more advanced cases. Suppers and attacks of dyspnœa stand in a significant relationship to each other. Often to take the first entails the latter; and both are retained or avoided at the discretion of the individual. A light supper, as a dish of oatmeal porridge, Revalenta Arabica, or lentil meal, or an oatmeal or arrowroot biscuit, with the fluids just mentioned, will often enable the patient to avoid a night attack of dyspnœa or asthma, especially if taken three or four hours before retiring to bed. If the patient feels hungry before the time for breakfast arrives, then a glass of milk, with a little grated nutmeg and a dessertspoonful of spirit in it, may be taken about five in the morning. Early in the day the bulk of food should be taken. The earlier in the day the food is taken the more probable is its digestion. It is well for the patient to lie

down after the noontide meal; if this is impracticable, then rest in an arm-chair should be essayed, and if a nap can be achieved all the better. A complex arm-chair for advanced heart cases has been devised by Dr. Dobell, and can be procured at most surgical instrument makers.

Next comes the question of alcohol in the treatment of heart disease. Some cases are benefited by alcohol, in others it certainly does harm. As a class, diseases of the heart do not require alcohol as part of their permanent treatment, until the final stages are reached. If it enables more food to be taken and digested, then it does good, and the amount required to attain this may be taken; but it should not be exceeded. Too much alcohol, whether in too large doses or in too frequent doses, is decidedly bad.

When, however, there is cardiac dyspnœa, and the right ventricle is overtaxed, then alcohol is indicated, and not only may, but even must, be given; and that, too, in no stinted quantities. In discussing the effects of alcohol in arresting or relieving intermittent pulse—commonly a serious indication of heart inability—even B. W. Richardson says: "If after great fatigue, or excitement, or anxiety, there is sleeplessness, restlessness, and painful knowledge on the part of the patient of the hesitation of the circulation, half an ounce or an ounce of brandy will act, generally, in the most effective manner. It will bring rest at once, and often, when a narcotic fails, sleep; but it must be repeated only after an interval of seven or eight hours; if it be carried to the extent of producing the third paralyzing or narcotic degree of alcoholic stimulation, it will have conferred evil instead of good" ("Discourses on Practical Physic," pp. 82, 83). Even larger quantities than those here spoken of may be required. We know that sleep depends upon—for one factor—an anæmic condition of the brain; also we know that sleep is often broken or prevented by imperfect action of the heart, the patient being wakened up with a start just when falling off asleep; alcohol improves the action of the heart, and does away with this painful start; while, by dilating the peripheral vessels, it does not induce a rise of the arterial blood-pressure—which would be inimical to sleep. The "night-cap" at bed-time has much to be urged in its favor; when the

contact of the cold sheets, sending a current of chilled blood from the cutaneous peripheral vessels to the heart, produces much pulmonary disturbance, as cough and dyspnœa, then it is well that the "nightcap" be taken hot when getting into bed; while the bed should be previously warmed. By these means combined, much night disturbance in heart disease may be avoided.

The clothing should be light but warm. The feeble circulation does not admit of a high body-temperature; consequently, it becomes necessary to conserve the body-heat to the utmost. Warm merino vests and drawers, flannel, lambswool, etc., should be worn. Weight for weight, undershirts are far before overcoats; the latter, when warm, are almost necessarily heavy, and tax the patient's powers too severely when taking exercise.

External measures, as belladonna plasters, are often serviceable. They almost always do good in palpitation. They certainly act well by impressing the mind, and are also physically useful. They may produce some of the physiological effects of belladonna, as disappearance of one iris almost complete. Such accidents usually occur with ladies, and create some unnecessary alarm. Blisters over the heart are troublesome, and their utility most questionable.

The German plan of wearing a flask of iced water over the heart may be useful in neurosal palpitation, from the sedative effects of cold upon the heart; but in organic disease rather a flask of hot water is indicated.

Exercise must be limited, and exertion forbidden. Carriage exercise, where the patient's means will permit of it, may be taken, even when advanced stages are reached. The bowels must be regulated, and no straining at stool must be permitted; death on the night-chair is not uncommon in heart disease. The bladder may require examination, and if involuntary dribbling exists a catheter must be passed; involuntary micturition is often the evidence of an overfull bladder, not of a flaccid one. This attention to the bladder is the more necessary, as prostatic enlargement is common in elderly men. Sometimes, too, the intellect is clouded, and the brain, sparsely fed with arterial blood and enfeebled by venous congestion, works but inefficiently, and the patient's habits and ways become altered, and he may become

inattentive and careless about his evacuations. Such are the measures to be taken and the points to be attended to, all through the course of chronic disease of the heart.

Treatment of Sequelæ.—This last division of the subject of the treatment of heart disease is of scarcely less importance than the previous ones. It can only alleviate; they may prevent or arrest disease. But the power to alleviate the many sore troubles which are the outcome of organic changes in the heart, is no small matter in practice. We have seen how we may increase the amount of blood in the arteries by exciting more energetic action in the heart; we now come to consider the various means of relieving venous congestion. Venous fulness is the cause of many disturbances. It leads to the growth of connective tissue in the liver, spleen, kidneys, and brain. It leads to albuminuria, to albumicholie (Oppolzer), and to gastric catarrh, as well as to effusion into the various venous sacs from the peritoneum to the ventricles of the brain. A frequent and troublesome instance of this venous congestion is bronchorrhœa, from congestion of the bronchial veins. Effusion into the subcutaneous areolar tissue, especially of the lower limbs, is one of the commonest and best known consequences of cardiac disease. By the fall and increase of the dropsy do the friends of the patient usually estimate the efficiency of the treatment adopted. If the dropsy increase while the patient is under energetic treatment the look-out is very dark indeed. Then there are troubles connected with the pelvic organs, enlargement of the prostate, vesical catarrh in men; menorrhagic losses and leucorrhœa in women; the first requiring buchu or pareira brava, the latter ergot, and tonics with the use of the bidet and the vaginal douche.

From the various maladies which are the results of venous congestion, relief may be afforded in several ways. The chief indication—the factor ever to be recognized, no matter what the adjuncts each individual case may require—is to unload the venous fulness. In doing so we achieve two ends: (1), we relieve the venous congestion, and, (2), we reduce the bulk of blood by the removal of a quantity of water. The blood having lost so much of its water, takes up more water, by osmosis, from the anasarcous limbs; and so by persistently, at brief intervals, tak-

ing away a quantity of water from the blood, we aid in draining the water-laden tissues. Purgatives are then of great value. In many cases the system spontaneously, and of itself, institutes diarrhœa, and in dropsy this must not be rashly arrested; often it is a most useful drain. (For the purpose of convenience here, dropsy will be used to illustrate the consequences of cardiac failure; but the remarks on the treatment of venous congestion apply to the other consequential results as well as dropsy.) Where the diarrhœa is natural, it must be left alone or even encouraged, unless the patient is distinctly suffering therefrom; and then a powder containing myrrh, in powder, and nitrate of bismuth, each ten grains, may be given at bedtime, with or without a grain of opium. Even a pill of sulphate of copper and opium may be required; but the diarrhœa must not be stopped until and unless some other water-emunctory be opened. When a purgative has to be selected to relieve the venous congestion, then one of the group of hydragogue cathartics must be chosen; mere laxatives here are futile and useless. H. C. Wood advocates one-sixth ($\frac{1}{6}$) of a grain of elaterium. I have found one-twelfth ($\frac{1}{12}$) of a grain with pulv. jalapæ co. ʒss., or pulv. scam. co. ʒss., very effectual. Compound jalap powder is in common use. Dr. Dowse, who had much experience in the Highgate Sick Asylum, prefers the compound scammony powder. Sir Robert Christison approves of gamboge, two or three grains rubbed up with a couple of drachms of bitartrate of potash. This is an excellent form; and if ten grains of black-pepper are rubbed up with the other two ingredients, all griping is usually done away with. Carminatives in full doses relieve griping from purgative medicines, whether in the ordinary laxative in common use, or in more active agents employed on special occasions. At first sight it may appear that active purgation will be ill-borne by patients with heart failure, but, in fact, they not only tolerate it well, but feel the better of it. Four or five free evacuations would depress an ordinary person, but the patient with hydræmia feels the lighter and better therefrom. The blood so depurated is improved in quality, and the patient feels enlivened and sprightly after. All drains set up artificially, act more potently after the effects of digitalis upon the heart have become apparent. There is no practical fear of syn-

cope or collapse from free purgation, after digitalis has been given two or three days, and the heart brought under its influence. Still, if any fear should exist, the nurse can be advised to have a little alcohol or sal volatile, with ether, at hand, in case the patient feel faint. Sometimes the cathartic acts inefficiently, and then it is desirable to combine two such agents. A grain of gamboge added to the jalap or scammony powders, will often increase the efficiency of the dose very materially. Purgatives are often stated to have a diuretic effect in cardiac dropsy; this is my own experience. The relief to the venous congestion is followed by a freer circulation through the kidneys, and, consequently, there is a better diuresis. Especially in combined heart and kidney dropsy are the good effects of carthartics well manifested, and the impression is strong on the writer's mind, that in several cases where the patients would not be purged, the fatal result which quickly ensued might have been averted, for some time at least, if the patients' objections to cathartics could have been overcome.

Diuretics are agents which increase the bulk of urine, and it is in dropsy they have mostly been tested, and where their reputation has been attained. Diuretics are of two kinds: (1), those that increase the bulk of urine solids, as potash, colchicum, and buchu; and, (2), those which increase the bulk of urine, as digitalis, belladonna, squill, scoparium, and other less known agents. The latter class, of which digitalis is the type, is the class from which we must select our remedial agents in dropsy. Digitalis, given to healthy persons in toxic doses, does not increase the bulk of urine (Winogradoff, Lauder Brunton), but lessens it. In digitalis poisoning there is suppression of urine (Christison). But in dropsy digitalis, by raising the blood-pressure in the arteries, and so increasing the blood-pressure on the glomeruli of the kidney, increases the bulk of urine very markedly. The different measures which relieve venous congestion affect the renal circulation. Thus, purgatives lessen venous congestion, and by diminishing the congestion in the renal veins, allow of better circulation through the renal vessels, and so of freer secretion of urine. As the blood-pressure in the arteries rises, the venous congestion falls. Consequently, a combination of measures is indicated. While the diuretic which acts through

the vascular system, and increases the bulk of urine by raising the arterial tension, is the agent we require in dropsy—as digitalis, it may be given with the other form of diuretic which increases the amount of urine—solids excreted—as buchu as a vehicle. Hammond, in an essay "On the Action of Certain Vegetable Diuretics," 1859, has pointed out the two classes of diuretics with much perspicuity, and Americans use diuretics more intelligently than we do.

Diaphoretics may be used with great advantage in dropsy. They consist of two divisions, (1), medicines, and (2), baths. Of medicinal diaphoretics, opium and antimony are out of place here. Stimulant diaphoretics, acetate of ammonia with nitric ether, or guaiacum, or ipecacuan, rather are indicated. But the condition of the stomach, or the general state, may forbid their use. Potash salts are all diaphoretic as well as diuretic, especially iodide of potassium, which may be given with guaiac or serpentaria. They may form part of the general treatment without disturbing the system; but for the production of acute diaphoresis it is well to resort to external applications. Even jaborandi, which promises to be a very efficient diaphoretic, may not be so safe and free from troubles as the bath. All baths adopted for the relief of dropsy should allow the head to be free and the respiration unimpeded; consequently, there are objections to the use of the Turkish bath, which, however, has its firm adherents. The vapor bath, the sitz bath, and the ordinary warm bath, are good. The most efficient bath, which can be used in the humblest homes, is that devised by Sir James Simpson. It consists of six or eight lemonade bottles filled with boiling water and tightly corked. Then a woollen stocking wrung out of hot water should be drawn over each bottle; the bottles should then be packed round the patient in bed. In fifteen minutes the patient will usually begin to perspire profusely, and this will go on for an hour or so. Then the bottles can be removed, with the blanket immediately under the patient, who is left to perspire steadily. Sleep often follows the sweating, which indicates the relief that has been afforded. In practice it is well to place the dropsical patient under a course of digitalis, with carbonate of ammonia and strychnia, or with iron; and then use purgatives and the

bath on alternate days; or, if that be too much for the patient, each twice a week, alternately. While seeking to relieve the patient the powers must be husbanded; and the active treatment, suitable in one case, may not be suited to another. In some cases it may be enough to give relief, while in another cure of the dropsy may be essayed at once and energetically.

The following case illustrates well what may be done by well-devised treatment in certain cases. The physique of the patient encouraged the energetic treatment adopted; in other cases it may be well to make the treatment less energetic, and be satisfied with a less rapid progress. On January 25th, 1877, J. H., æt. 69, a brewery man, was admitted into the West London Hospital under my care. He was a tall, stalwart man, with white hair and a florid complexion; of grand physique, and of a distinctly gouty diathesis. Has always been a very temperate man. General health always been excellent, except occasional attacks of rheumatism. Has lost all his teeth, which came out without a speck of disease on them. The radial artery is somewhat atheromatous, while the left temporal artery is quite calcareous. He had at this time extensive dropsy. There was considerable œdema in the lower limbs, extending up to the groin, but without effusion into the scrotum. The knee joints were greatly distended by fluid. The upper limbs were more swollen than the lower limbs. On making firm pressure over the brachial arteries, so as to press through the effusion, a distinct thrill could be felt, most marked in the left brachial. The area of cardiac dulness was quite resonant from emphysema. The heart's apex could be felt in the seventh interspace. The first sound can there be heard of normal character. A systolic murmur can be heard at the base; and the aortic second sound is muffled. P. 100, with slight irregularity. This pointed to mitral disease, but no mitral murmur could be detected. Urine very scanty, and full of lithates, only half a pint being passed in the first twenty-four hours. No albumen.

Diagnosis, combined heart and kidney dropsy.

Prognosis, favorable as to relief.

Treatment.—To have a purgative and a vapor bath on alter-

nate days, with a mixture of potas. iod., gr. v. Pot. bicarb., gr. x. Inf. digitalis, ʒj. Inf. buchu, ℥j, ter in die. Milk diet.

January 26th.—Had slept a little. Had a purgative powder—elaterium, gr. $\frac{1}{12}$. Pulv. jalapi co., ℈ij, at 5 A.M. Had four motions by 10.30 A.M. Tongue dirty at base and middle, red at tip and edges.

27th.—Had a vapor bath, but did not sweat.

28th.—Decidedly better. Tongue clean; swelling of arms and legs much reduced. Urine acid, sp. gr. 10.20, clear; still but half a pint.

29th.—Dropsy falling steadily; a little return in right arm. The thrill in the brachial arteries is not nearly so distinct. Some irregularity of pulse still. No systolic bruit at base to be heard, but a systolic mitral bruit can be heard distinctly, an inch and a half to left of left nipple in the seventh intercostal space, P. 104. Powder and bath continued.

30th.—Altogether better. Urine ℥xij.

February 3d.—Sleeping well; appetite good. Heart-sounds as on 29th. In consequence of the presence of the mitral lesion the dose of digitalis was increased. Pot. bicarb., gr. xv. Ferri am. cit., gr. v. Tinct. digitalis, ♏x. Inf. buchu, ℥j, ter in die. The powder twice a week, and the bath twice a week. Some fish in addition to the milk diet.

Condition much improved. Dropsy nearly gone. Effusion into knee joints entirely disappeared.

5th.—Irregularity in pulse quite gone. Pulse good. Tongue clean. Passed three pints of urine, clear, non-albuminous.

8th.—Thrill in brachial arteries gone. Slept well; felt well. Passed three quarts of water. Mixture as before. Powders and bath to be discontinued. A little meat and a pint of beer to dinner. To get up a little.

From this time the patient continued to improve steadily. The dropsy disappeared entirely. The pulse is regular. The thrill in the brachial artery can be felt occasionally. The patient gets up every day.

17th.—The house surgeon (Octavius White) has written "Discharged. Cured,"—a very sanguine view of the case.

The old man has since attended as an out-patient, and takes a

mixture containing digitalis. He is a very hale, straight old man, and walks his two miles to the hospital without distress. A systolic mitral murmur at the left apex can usually be heard, but faint and low when audible. There appears no tendency in his mitral disease to progress, or if so, very slowly.

Remarks.—This case is interesting from its *tout ensemble* being that of combined heart and kidney dropsy; a view in no way negatived by the absence of albuminuria. But the mitral complication gave it peculiar characters. The rapidity of the pulse, and the compressibility of the radial artery, though atheromatous, pointed to a mitral lesion; even when no mitral murmur could be found. The small bulk of urine laden with lithates also suggested a mitral lesion, a view favored by the irregularity in the pulse.

The increase in the bulk of urine under the treatment is suggestive. Until the presence of the mitral lesion was testified to by the presence of a murmur, the dose of digitalis given was small. Afterwards it was increased with the following results as regards bulk of urine. From January 25th to the 29th the amount passed was but half a pint. On the 30th it was twelve ounces. On February 1st, 2d, and 3d it was one pint. On the 4th the dose of digitalis was increased from ℥j of the infusion to ten drops of the tincture. It took some time for the agent to act on the arterial tension, and though the dropsy kept falling there was little increase in the bulk of urine passed. On the 6th it had risen to six pints, at which it continued until his discharge on the 17th. It has remained about this ever since December, 1878. The influence once attained was maintained by the digitalis, but it took a little time for it to tighten down the heart and arterioles on the blood-column, and so increase the blood-pressure on the glomeruli of the kidney. It is in this way that digitalis is a diuretic. When taken by persons in health it soon lessens the bulk of urine, as was found by Stadion and Winogradoff.

Another remedial measure is puncture. This may be done with a needle or with a lancet. Sometimes the flow so induced is very copious, and the relief afforded is great. In my own experience I have seen little good come from punctures in cases of pure cardiac dropsy; but where there has been a well-marked

renal factor in the case, this puncture has usually been very useful. The use of the needle is sometimes followed by spirts of fluid, until the bed is sopping wet. To obviate the last, Dr. R. Southey has devised a most useful instrument. It consists of a hollow needle, like the needle of a hypodermic injection syringe, to which is attached an india-rubber tube. These needles are inserted into the areolar tissue under the skin, while the distal ends of the tubes are placed in a suitable receptacle. All wetting of the bed is thus avoided, while, as a means of drainage, this plan is very efficient.

At times diminution of the dropsy will occur spontaneously. When this is synchronous with a sharp diarrhœa it is well; but when it is the result of œdema of the lungs or effusion into the serous cavities, then the disappearance of the anasarca is not a matter for congratulation.

There are still one or two points to be attended to in the treatment of chronic heart disease. One is *heart-cough*. This is due to congestion of the pulmonary circulation. It is a dry, short cough, usually without expectoration. It is very common in mitral disease in young subjects. The great matter of importance in practice is not to give any opiate cough medicine for heart-cough. Opium relieves the cough, but aggravates the general condition. Most lamentable results have followed the use, or rather abuse, of opium in heart-cough. The cough is one of the consequential outcomes of heart failure, and is most effectually relieved by improving the general condition of the vascular system. Carbonate of ammonia and digitalis are the remedies *par excellence* for heart-cough.

Then as to pain at the heart. Pain at the heart is usually neuralgia of the sixth or seventh intercostal nerves. In intercostal neuralgia there are usually three very painful spots—the tender spots of Valleix—one, the most frequent just at the left apex; a second at the base of the left scapula, and the third where the nerve issues from the vertebral column. This pain is in no way related to the heart. Then there is another pain felt over the base of the heart. It is usually distinctly in the thoracic walls, often cutaneous. It is found with phthisis as much or more than with heart disease. What is its relation to disease of the heart, even if the pain is in any way connected with the heart, is

as yet utterly unknown. The strong probability seems that it has nothing to do with the heart.

Intercurrent disease of all kinds is very serious in a person suffering from organic disease of the heart. Bronchitis is especially dangerous. It must be met by the most stimulating expectorants; and digitalis, to keep up the action of the left ventricle, must be freely exhibited. A shrewd old practitioner in the north of England once informed me—when expressing my chagrin at losing a patient in bronchitis who had old-standing mitral disease, whom I thought ought to have recovered—that all such patients would die, I should find. This was not encouraging; but, reflecting on the remark, it became evident that there must be some factor not recognized in the treatment, and the most probable thing was the effect of the heart-lesion. Afterwards this factor was allowed for, and digitalis was freely given in such cases in future with the most gratifying results. Probably, in all intercurrent disease, it is well to bear in mind the condition of the heart in all plans of treatment. Even trouble in another organ affects the heart, and Botkin, of St. Petersburg, tells of a case where a floating kidney was the source of much cardiac disturbance.

Heart Diseases in Children.—Until recently heart disease in children was little attended to unless cyanosis was present. We now know that mitral disease is common, especially in little girls. It is apparently a common consequence of scarlatina, as well as of acute rheumatism. Its detection is unusually easy, provided the heart be auscultated. Very commonly mitral disease affects the growth, the children being small and puny. At other times there is no evident ill effect produced by it. Even shortness of breath on exertion is not noticeable. There is a great tendency for the valve mischief to remain static in children. Many cases I have followed for years; one or two through puberty to manhood and womanhood. A number break down at puberty; others get over this time very nicely. In one case I am awaiting the results of marriage with much interest.

The treatment consists of the administration of digitalis steadily and continuously. My favorite combination is tincture of digitalis, five drops, potassio-tartrate of iron, five grains, three times

a day, for months. This usually produces much improvement. During periods of acute growth it is very desirable that the little patient be put to bed. Heart-cough is in children generally a good informant of their state; when getting worse, the cough is more troublesome. Good, easily digestible food is indicated, with a moderate amount of exercise. As a rule, the presence of mitral disease does not disqualify the child from attendance at school, nor cripple its intellectual development. This is fortunate, for such children are incapacitated from entering toilsome occupations, and must live by the use of their brain, and are only fit for light occupations. Acquired valvular disease does not cripple the organism like congenital disease with cyanosis.

Finally, every case of heart disease is a subtle problem to be solved; whether it is in that stage when recovery or repair is possible, or in that more advanced condition where all treatment is but palliative, or even a means towards euthanasia.

CHAPTER XIII.

PERICARDITIS—ACUTE—CHRONIC—PERICARDIAL ADHESION—HYDROPERICARDIUM, ETC.

THE pericardium is the serous membrane which envelops the heart. It consists of two layers, the internal, or cardiac layer, and the external, or outer layer. By means of these two serous layers the movements of the heart are carried on with the least possible friction. The internal layer covers the heart; it extends upwards, nearly to the top of the aortic arch, when it is folded back, forming the outer layer. This serous sac may contain a small amount of fluid in health. In some persons the pericardium is congenitally wanting; but there are no means of ascertaining this in life. Like all other serous sacs, the pericardium is liable to inflammation. Pericarditis is not common except as an accompaniment of rheumatic fever. Sibson found that in 326 cases of acute rheumatism admitted into St. Mary's Hospital, one-fifth (63) had pericarditis, which was accompanied in 54 of them by endocarditis; and only in one-fourth of them (79) was there neither pericarditis nor endocarditis. One-third of the whole cases (108) had endocarditis (simple); and a fourth (76) had threatened endocarditis, the signs being transient or imperfect.

Acute Pericarditis.—This malady, though usually found along with rheumatic fever, may be the result of cold, of injury, of gout, or erysipelas, or in the course of the exanthemata, from an eruption of pus, or phlebitis (Hayden). When the pericardium first begins to inflame, it presents an appearance of general redness, with ecchymosed spots, often of a dendritic character. There is swelling in the serous and subserous coats, and the formation of delicate villous projections, with effusion of a serous fluid, usually containing shreds and filaments of lymph. This fluid varies in amount and quality. Frequently it is considerable in amount and distinctly fluid; at other times it is more decidedly albumin-

ous, and so dense as almost to possess consistency, so that the two layers are adherent, and when separated present a honeycombed appearance strongly resembling "tripe," or two buttered surfaces, which have been applied to each other, and then been pulled asunder. At other times, when associated with dyscrasial affections, the exudation is bloody, and then the term "hæmorrhagic" is applied to the pericarditis.

"When the surface of the heart becomes inflamed, a blush of fine vessels, consisting of a velvety network, appears on the surface of the organ, and especially over the larger coronary vessels at the base and septum of the ventricles. The inner surface of the pericardial sac, wherever it rests upon the inflamed heart, kindles also into a blush of fine vessels. The inflammation caught from the heart on the inner lining of the sac, spreads rapidly to the fibrous structure of the pericardium, and through it may even often extend to the surface of the pleura covering the sac. The inflammation of these parts tells upon the nerves distributed to them. The surfaces of the heart and sac, instead of being smooth and glistening, become dull and velvety; and fluid is poured out and lymph exudes from the inflamed surfaces. The liquid in the pericardium increases rapidly. At first it falls into the back part of the sac, but as it increases in quantity it makes a space for itself between the floor of the pericardium, which it depresses, and the lower surface of the heart, which it elevates; and it gradually distends the pouch in every direction, displacing the lungs to each side in front, pushing the central tendon of the diaphragm, the stomach, and the liver downwards; and pressing backwards, when the distension from the fluid is great, upon the bifurcation of the trachea, the left bronchus, the œsophagus, and the aorta" (Sibson). The fluid also compresses the heart and the veins and arteries contained within the internal sac.

Pericarditis has several terminations. Absorption may take place and extend to a *restitutio in integrum*. While the fluid is absorbed in some cases, fibrinous shreds form bands which bind the two pericardial surfaces together; these are dragged upon by the heart's movements, and form in time fibrinous bands like those found in pleuritic adhesions; at other times the adhesion is more general, until it may even obliterate the pericardial cavity.

The effused fluid may undergo changes and become purulent, and resemble the contents of the pleuritic cavity in empyema. Perforation may occur through the chest-walls, or into the lung, permitting of the admission of air into the sac, forming pneumopericardium with ichorous pus. Most commonly the fluid contents are absorbed, the more solid ones remaining. These inflammatory products may undergo fatty degeneration, and remain as cheesy or mortar-like masses; or the fluid portion of the degenerate mass may be absorbed, leaving chalky masses behind; or the connective-tissue corpuscles may become more or less organized, with a deposit of lime salts in them, and an ossification, or rather petrification, may result. This may be so complete that the heart may be inclosed in a calcified envelope, instead of its serous coverings.

The pericardium may, like other serous coverings, become the seat of gray or miliary tubercle, and be studded therewith. Even the larger masses of yellow tubercle have been found on it.

In connection with pericarditis must be mentioned "milk spots," or "white patches," which were at one time thought of some importance other than pathological; they were supposed, from the frequency with which they were found in soldiers, to result from the "cross-straps" which traverse the region of the heart, and were accredited with having something to do with death from heart disease. They are now known to be very innocent growths of white connective tissue immediately beneath the cardiac pericardum, utterly free from any malign influence upon the tissues underneath them. The epithelium is perfect on the true "milk-spot" (Hayden). In size they are about half an inch across, and in thickness vary from half a line to two lines.

Pericarditis is usually accompanied by more or less extension of the inflammatory process to the muscular structure of the heart. This is usually superficial only; but at times it extends more deeply, and produces softening of the heart-fibres. If there be also endocarditis extending into the muscular structures, the heart-fibres may yield and dilatation be the consequence. This may result from the heart-fibres being weakened by infiltration of serum, without any true carditis having taken place. Very commonly pericarditis is accompanied by endocarditis; but they are

not results of each other so much as the results of some exciting force causally common to each. The external layer of the pericardium may become affected by continuity from pleurisy, or even pneumonia.

Oppolzer divided pericarditis into four divisions:

1. Idiopathic, or simple, where it is unconnected with constitutional conditions; as where it is traumatic in origin.

2. Consecutive, where it follows pleurisy, disease of the sternum or ribs, tubercular masses in the lungs, etc.

3. Symptomatic, where it is part of a general condition, as rheumatic fever, Bright's disease, scarlatina, syphilis, etc.; and

4. Metastatic, as in septic conditions, pyæmia, or puerperal fever.

Pericarditis is frequently associated with scurvy and purpura. It has been met with in Russia as an epidemic, and is often seen in this form in attacks of scurvy along the shores of the Baltic (von Dusch).

But as usually seen, pericarditis is found in connection with rheumatic fever; consequently, its symptoms are obscured or masked by those connected with the general condition. Even the pain in the chest may not be noted by the patient, the attention being drawn to the more acute pains felt in the joints. Commonly there are rigors at its onset, but these may be wanting. The patient is usually flat on the back, an attitude due probably rather to the joint affection than to the pericarditis. There is a plaintive look of suffering and helplessness, with desire for succor, on the patient's features. The pulse at first is rapid and rather full. "The action of the heart is accelerated, the impulse is sharp and abrupt, and the sounds, especially the second, are clear and ringing, with the addition of a characteristic rustle, distinct from friction-sound" (Hayden). But this soon gives way to a weak, rapid, compressible pulse, occasionally very rapid, at other times becoming abnormally slow. There may be palpitation and considerable excitement in the early stages, but they soon give way to evidences of cardiac debility. There is also headache, dizziness, and tendency to delirium at nights; in some cases there is very active delirium, especially at nights. It is difficult to state the temperature—what is due to the pericarditis,

and what is due to the rheumatic fever, but it usually does not exceed 104°. In the later stages the temperature may fall below the norm, especially in the fatal cases. There is often gastric derangement, probably from the connected nerve supply. The urine is small in quantity, usually laden with lithates, and often free from chlorides, and this, too, where there is no lung-complication. It is not rarely albuminous.

From the absence of any pathognomonic symptoms or signs—for the pain (intensified by pressure) may be wanting, or overlooked amidst the general condition of pain, it is well to examine the heart daily in cases of acute rheumatism; but the examination should invariably be conducted with the greatest care; the chest should be exposed as little as possible and for the briefest practicable period; indeed, there should be but a hole in the flannels through which a warmed stethoscope should be passed. To inspection there is nothing until the stage of effusion is reached, when there is bulging in young persons whose costal cartilages are still flexible. On palpation the excitement of the first sound may be felt; and as soon as there is friction, betwixt the two dry, inflamed, and, later on, roughened surfaces; this can be felt. We can feel that friction which is communicated to the ear as a murmur. This sensation of friction is lost as effusion takes place; not only so, but the heart's impulse becomes faint and imperceptible.

Percussion tells us nothing until the stage of effusion is reached. The evidence furnished by it is negative until there is effusion, which, when it occurs, gravitates to the back of the pericardium, and then gathers round the root of the aorta, where it may be mistaken for a tumor or aneurism. As the distension proceeds, the pericardium assumes the shape of a rude triangle. The dulness is broadest in the recumbent posture, and is diminished laterally on the patient sitting up. The triangular shape of pericardial effusion, the narrowest point being uppermost, distinguishes it from the dulness of hypertrophy, especially right-side hypertrophy. In right-side hypertrophy, the dulness never extends to the left of the left apex, while in pericardial effusion the dulness extends beyond the normal point of the left apex. In left-side hypertrophy the strong apex beat is felt away to the left of its normal seat.

"During the stage of absorption, the apex returns to its normal position; the dulness gradually disappears; the sounds and the impulse regain more of their normal character, the friction reappears and then ceases." (Da Costa.)

When the effusion into the pericardial sac is very large, it may produce dulness at the back; otherwise the clear note at the back of the chest serves to distinguish pericardial from pleuritic effusions.

Auscultation is the means of examination which furnishes us with our most trustworthy information as to the existence and extent of pericarditis. The two dry inflamed serous surfaces produce a friction murmur, which in loudness and extent of area is in proportion to the extent of the inflamed surface. Thus it may only be heard over a limited space where a small part of the pericardium is affected; while in other cases it is heard over the whole cardiac area. It has been described as a "to-and-fro" sound,

FIG. 41.

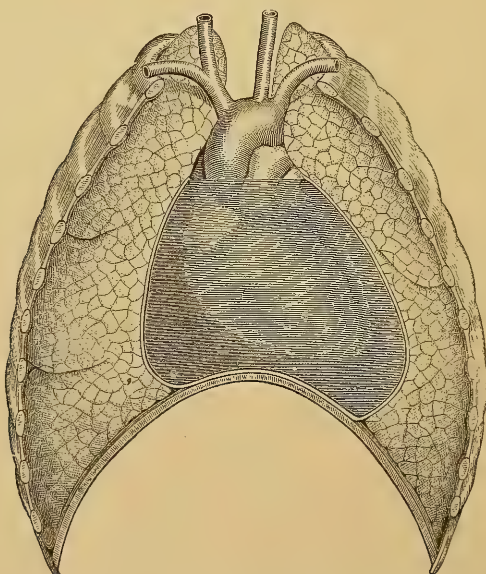


Illustration of the position of the heart in pericarditis, and of the distension of the pericardium with fluid. The heart sounds are indistinct, excepting above the effusion; the impulse is feeble. The extent and shape of the percussion dulness may be judged of by the appearance of the distended sac.—(From Da Costa.)

corresponding to the movement of the heart in systole and diastole (see p. 29); but the late Hyde Salter described it as being a triple sound, a canter—in fact a "rub-a-dub-dub," the sounds being produced (1) by the auricular contraction; (2) by the ventricular systole; and (3) the sound produced by the diastole. In this he is followed by Professor George Johnson. Pericardial friction-sound is a subject which may be discussed at great length, but it is questionable whether such discussion would be very profitable. Suffice it to say that the sound may be heard more or less extensively in different cases, and may soon disappear, or, in severe cases, remain till the surfaces are separated by effusion. The main point on which stress may be laid, is the distinction betwixt an exocardial and an endocardial murmur.

In pure cases of pericarditis and endocarditis the distinction may be made out; but as the two are very commonly found together, the diagnosis is often physically impossible.

The distinction in well-marked cases is given by Da Costa as follows:

ENDOCARDITIS.	PERICARDITIS.
Blowing sound; excited action of heart.	Friction-sound; excited action of heart.
Slight, if any, increase of percussion dulness.	In stage of effusion, marked and extended percussion dulness.
Impulse strong.	Impulse wavy and feeble.
Sounds normal, or more distinct, except at part where the murmur is heard.	Sounds feeble and muffled; no blowing sounds.

In pericarditis the sounds are not heard at and conveyed away from the orifices, as in the case in endocarditis. If the sound be heard on the right side, it is probably pericardial, as endocardial murmurs are usually heard on the left side, being mitral, and but rarely aortic. When the aortic orifice is implicated in endocarditis, the sound is conveyed along the aorta forwards. Dr. King Chambers, in his "Clinical Lectures," advocates a plan of discriminating betwixt endocardial and exocardial murmurs as follows: Apply the stethoscope as usual, and then put the ear to it; having found the murmur gradually withdraw the ear, the instrument being retained *in situ*. If the murmur remain as long as the heart-sounds are audible, the murmur is endocardial; if the murmur is

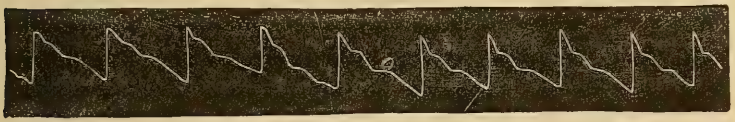
lost while the heart-sounds are still audible, the murmur is pericardial. This is ingenious, but it needs further corroboration.

At times a difficulty arises as to whether the friction-sound is pericardial or pleural. This can usually be determined by making the patient hold his breath. At times "the friction may be engendered in the pleura, and be caused by the movements of the heart." (Da Costa.) This authority says these niceties of diagnosis are comparatively unimportant to the treatment, which is the same in peri- and endocarditis till the stage of effusion is reached.

The *pulsus paradoxus* has been described by Koussmaul and Griesinger, and in this country by Dr. Pearson Irvine. The phenomena met with are "slowing" and "lowering" of the pulse with every inspiration; these being easily perceived in many cases by the unaided finger. They are met with in cases of adherent pericardium and pleura—a condition which enables the inspiring lung to draw on the (probably already weak) ventricle and interfere with or retard its action. Dr. Irvine has pointed out that forced inspiration will manifest the phenomena in a marked degree, and has described several cases in which, with the *pulsus paradoxus*, there were developed the usual probable signs of pericardial with pleuritic adhesions. In one instance the pulse with deep inspiration disappeared entirely from beneath the finger, and, on its return, was much retarded. In others the pulse became running in character, so that each beat could scarcely be separated from its fellows. All these are indications of a weak and laboring ventricle, yet, as Traube has urged, they do not occur in simple dilatation, nor in simple pericardial effusions, though in both the ventricle is impeded in its action. The diagram given below is that of a pulse-tracing taken in one of Dr. Irvine's cases. The pericarditis and adhesion developed in hospital, and with evidence of them the change in the pulse was noticed. It will be seen that the diagram shows slight depression with ordinary inspiration, that with the commencement of prolonged deep inspiration the pulse was lowered and the curve distinctly modified, and during the continued forced inspiration the pulse was slower and betrayed a laboring ventricle. The conditions could be readily perceived by the finger, and their de-

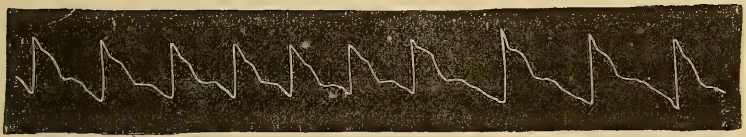
termination is all the more valuable because it can be made without instruments. The therapeutic value of observing this *pulsus paradoxus* is evident.

FIG. 42.



Ordinary Respiration.

FIG. 43.



Prolonged Inspiration.

FIG. 44.

Continued Inspiration.
The pressure is the same in all, viz., 5 oz.

According to Stokes, pericarditis may be circumscribed either by a limited production of lymph, where the friction-sound is only heard over a limited area; or after pericarditis there may be almost general adhesion and a friction murmur only be heard at certain points, generally at the base, and this may remain for a long time. Hayden gives such a case, where the sound was restricted to the root of the pulmonary artery. "It was remarkably persistent," he says.

There is acceleration of the pulse and respiration, the latter being due, according to Sibson, to (1) the inflammation of the heart; (2) to distension of the pericardial sac pressing against

the diaphragm; and (3) the supervention of pleurisy. Sometimes the difficulty of breathing is such as to necessitate the patient's being propped up in bed, it being impossible to remain in the recumbent posture. The excitement produced by the inflamed surfaces at first, causes more rapid action of the heart; while when there is effusion it presses upon the heart, interfering with its being filled in diastole, and thus maintains rapid action of it; or, at times, slowness and irregularity. How far the cardiac nerves are affected in pericarditis, it is difficult to say; pressure on the phrenics, and especially the right phrenic, produces the persistent hiccough not unfrequently found; indeed, it may go so far as to produce paralysis of the diaphragm, with falling of the abdomen on inspiration. When this occurs there is great depression.

Acute gastric symptoms are sometimes set up by pericarditis, where we must suppose the vagus to be the means by which this is brought about. Here the symptoms of mischief within the chest are latent and in abeyance, and there are nausea and vomiting, "fever, dry red tongue, extreme epigastric, and no præcordial tenderness, bilious vomiting, and diarrhœa, and perfectly regular pulse—present not a single symptom connected with the heart" (Walshe).

Sometimes pericarditis simulates acute meningitis. This is of the more moment in that rheumatic metastases to the brain are not unknown, and are unfortunately almost invariably fatal. While the wild delirium set up by pericarditis suggesting acute inflammation of the brain or its meninges, is usually free from serious results. That pericarditis should set up acute brain symptoms is probably due to the fact that it occurs in conditions of poisoned blood, whether in acute rheumatism, Bright's disease, or septicæmia. Austin Flint has given careful attention to the head symptoms set up by pericarditis, and gives two cases in detail. Both were typical cases of pericarditis simulating meningitis, and in both he observed a peculiarity in the delirium. This was the fixed impression of having committed a crime. He says, "a fixed delusion of having committed some crime appears to be a distinguishing feature" ("Diseases of the Heart," p. 358).

The prognosis varies with the associations of the pericarditis.

Rheumatic fever, whatever its complications, is but rarely fatal. Where the pericarditis is severe and accompanied by considerable endocarditis, Walshe thinks the prognosis more serious than when the pericarditis is single. The nature of the effused fluid will influence the prognosis; thus whenever pericarditis is found along with dyscrasial conditions it is serious; and that, too, without distinct septicæmia being present. The mere amount of pericardial inflammation does not materially influence the prognosis; but the amount of subsequent pericardial effusion does. And if the effused fluid be hæmorrhagic or purulent, then the aspect of the case is serious. According to Stokes the prognosis in pericarditis may change suddenly, and great watchfulness should be exercised in such case.

The termination of pericarditis is varied. The fluid may be absorbed until no trace of the malady remains. More frequently the shreds of lymph form bands, or a more general adhesion may result, termed "adherent pericardium," which will be discussed in a later section of the chapter; or the fluid may remain in whole or in part, and may become puriform and open, like an abscess; or again, the fluid may be absorbed, and the solid matter remain.

The treatment of pericarditis is a very difficult subject to discuss on paper; at the bedside in each case the task is easier. Of old they bled heroically; then came the more modern school, where moderate bleeding by leeches was followed by the administration of alcohol, as represented by Stokes and Hayden.

The names of Law, Graves, and John Taylor are associated with the free exhibition of mercury in sthenic inflammation, especially of serous surfaces; the mercury being held to modify the effused lymph, and promote its absorption. Then the German school advocates the use of cold applications. Arguments could be found, doubtless, in support of each view, and in different cases. A little dogmatism may be permissible under these circumstances. Seeing that pericarditis is usually associated with acute rheumatism, we must include the treatment of the general condition in the remarks here made. The patient should be put into a flannel night-dress, or if in humble life into the flannel shirt of some male of the family. The sheets should be removed, so that the

patient lies in blankets. The chest should be kept warm in flannel, and should be as little exposed in examination as is compatible with a fair understanding of the morbid changes going on. As soon as pain is complained of in the chest, a hot poultice should be applied to the præcordium; and, as Dr. Lauder Brunton insists, thought may be applied to the putting on of a linseed-meal poultice. If applied to the skin at once, he says, it will scald the patient; if kept to cool it is virtually largely wasted. Several layers of flannel should be placed over the chest first, and then the poultice may be applied perfectly hot without scalding. The poultices should be changed as seldom as possible. Where the patient cannot bear the weight of the poultice, the pressure increasing the pain, the chest should be carefully covered with cotton-wool. The cutaneous and outer pericardial arteries come from the common intercostal trunk, and to dilate the cutaneous branches is to drain the pericardial branches; that is the principle of the local treatment. Some authorities advocate leeches to the præcordium, or cupping, or blisters; but external heat achieves the end arrived at just as effectually. Bleeding from the arm had its advocates, and probably has still. I have only seen it tried once, and that was in the case of a servant of an old-fashioned doctor. On the first hint of a venesection, he eagerly got out his lancet, and bled to sixteen ounces. The effect upon the pulse was very marked; the hard, tense, incompressible pulse became soft; the pain was relieved, and a hot poultice completed the cure. In young subjects under similar circumstances bleeding is quite permissible.

But in the majority of cases the treatment must be less heroic. Even purgatives are to be given cautiously, as all movement aggravates the patient's condition, and entails exposure, which is to be so carefully avoided. Opiates to relieve the pain are clearly indicated, but must be carefully administered in great depression, owing to the palsying effect of opium upon the centres of the circulation and the respiration. If given as Graves pointed out, they should be administered about half an hour before the usual hour of sleep; so that the stage of excitement be over, and the narcotizing action be present when the habitual time of sleep comes on. By such means the maximum of good effects will be secured with

the minimum dose. Opium may be given with comparative freedom; it acts upon the peripheral endings of nerves; it influences vascular congestion, and it soothes the system generally; and renders the pain produced by the friction of the inflamed surfaces more tolerable. ("Practitioner's Handbook of Treatment," Chapter XIII, Action and Inaction.) Diaphoretics may be combined therewith as ipecacuanha in Dover's powder; or two or three grains of powdered guaiacum with a grain and a half of opium (for an adult man); or if the bowels be confined, the same dose of opium with from three to five grains of the Pulv. Aloe comp. When opium is given along with alkalies, the dose must be increased. ("Fuller on Rheumatism and Rheumatic Gout.") And where pericarditis is the accompaniment of acute rheumatism, the alkaline plan of treatment is a good one in practice.

But in so treating pericarditis it is well to remember that endocarditis is its common associate; and the reader may here refer to Chapter VI (pp. 146–151), where the lines of treatment to be pursued in the management of endocarditis are given. The presence of pericarditis, however, makes a certain amount of difference. The heart may be so embarrassed by the pericardial effusion, or by the adherent surfaces, where the effused material is highly albuminous, that digitalis, or belladonna, or strychnia may be indicated in order to prevent the heart stopping in diastole. The heart must be kept going; and the risk of increasing the tension on the auriculo-ventricular valve-curtains during systole must be run; the immediate danger is too great and imminent to be subordinated to ulterior considerations. Alcoholic stimulants may be absolutely indicated, and Stokes says, in speaking of the treatment of pericarditis: "In truth it may be said that no man is fit to treat general disease, or local inflammation, especially its secondary forms, until he has conquered that fear of stimulants, which a long course of erroneous teaching has instilled into his mind." Probably the use of the agents which excite more perfect ventricular contractions, of which digitalis is the type, will do away with much of the necessity for alcohol except in extreme conditions. The state of the pulse, the amount of prostration, the severity of the pain, the temperature, and the number of respira-

tions per minute, must guide the practitioner in his treatment of each case.

Niemeyer says, "Calomel and blue ointment, in spite of the praises of English physicians, are not only useless but hurtful." In return it may be said that the German plan of applying cold over an inflamed pericardium is not very happy in its results, if the writer's experience in the Pathological Institute of Vienna is to be trusted.

The treatment to be pursued is to adopt the *via media*. Not to let the patient sink at the time for want of tonic and stimulant treatment, through fear of inflicting avoidable injury on the mitral valve; and, at the same time, to avoid all unnecessary stimulation—bearing the ulterior effects of endocarditis in mind.

"It is not uncommon to have attacks of pericarditis in which there is but little effusion, and that little localized at the roots of the large vessels. The symptoms of such mischief are slight, and the physical signs not well marked, though the ultimate consequences of the complication, if it be neglected, are highly injurious. Friction is frequently not heard, especially in the case of patients not under daily observation; the heart's sounds are somewhat muffled and distant at the base; the heart's action is weak comparatively, and the apex is not displaced upwards, or only slightly so; but at the base there is a square-shaped dulness, reaching possibly the second cartilages, and passing from this level into the normal cardiac dulness. This dulness varies from day to day, and in favorable cases rapidly disappears entirely. The signs enumerated are to a certain extent characteristic when they are met with in persons who have recently passed through an attack of articular rheumatism, and they should always be looked for in persons suffering from an attack of ambulatory character. If the pericarditis is not discovered, and be consequently neglected, adhesions of the pericardium readily results, and an insignificant disease, which rest would have cured completely, ends in mischief almost as grave as severe valvular imperfection. In the cases alluded to the inflammatory products are lymph-like, and readily undergo organization into fibroid tissue" (Pearson Irvine).

Having got over the acute stage, next comes the question of the treatment of the effused fluid. As to local treatment, the appli-

cation of a blister has its advocates, and is stated to aid the natural attempts at absorption. Hayden is opposed to it, and says: "In the treatment of liquid collections in the pericardium I have no faith in blisters, and rely mainly upon diuretics and hydrogogue purgatives." While Sibson says: "Blisters, besides inflicting local injury, taint the blood by increasing its fibrin, and are apt to lead to a secondary and low kind of inflammation in distant parts, and perhaps even to degrade the character of the pericardial inflammation itself, and to prolong its existence." Free purgation, warm baths, and the exhibition of iodide of potassium in infusion of buchu, are the measures to be relied upon to procure absorption of the effused fluid.

Paracentesis pericardii, though only quite recently generally adopted, was advocated by Riolan, Senac, Laennec, Skoda, and others. Dr. Clifford Allbutt has recorded two cases where the operation was performed for him by Mr. Wheelhouse, who says, "I chose for my purpose a small trocar. This I placed on the upper margin of the fifth rib, half an inch to the left of the sternum, and inclining it upwards and inwards, thrust it steadily forwards through the intercostal space towards what I believe to be the centre of the ventricle. I pushed it onwards until I could distinctly feel the movements of the heart with the instrument; and then, sheathing the point, I advanced the canula well up to the heart, until I could feel and see, and demonstrate to those around, the impulse of the heart as communicated to the instrument. The trocar was then withdrawn and the fluid allowed to escape. This it did at first in a steady stream, which soon subsided to a saltatory flow coincident with the heart's contractions. The fluid consisted of a pale pink coagulable serum, and, upon the whole, about three ounces escaped. During the operation the patient gradually obtained relief, and after the canula was withdrawn, the bed rest was removed and he was able to lie down." The patient recovered completely. Dr. Sibson says: "Extensive incisions and irritating injections should be of course avoided." The operation requires care; and a case is on record where a dilated right ventricular chamber was tapped for the distended pericardium, as it was supposed.

In the pericarditis of dyscrasial conditions it is difficult to say

what should be done. The treatment of the pericarditis is involved in that of the general condition. Stimulants must be administered after the stage of effusion is established, but during the dry stage it is doubtful if any good they might do would counterbalance the increased rapidity of the heart-stroke, and the increased friction their administration would almost inevitably induce.

Chronic pericardial effusion may produce symptoms very similar to those of angina pectoris. Dr. Caleb Hillier Parry, in an essay on Angina Pectoris, published in 1799, quotes such a case from Morgagni: "A Bolognese nun, while sitting up in bed and dressing herself, was suddenly seized with a sort of oppression about the heart, and subsequent fainting. From that time the oppression never ceased to be aggravated whenever she spoke or moved too much. The color of her face was good; her sleep undisturbed; her bowels and menstrua natural; her respiration equally easy whether she stood or lay on her back, or either side; her pulse neither tense nor hard, nor vibrating, nor in any respect unequal; no palpitation or great pulsation in the breast; no pain in the region of the lungs. Albertini (who had pronounced the disease to be a dropsy of the pericardium) formed his opinion from the following circumstances: that the patient felt her heart to be loaded, as it were with a rock placed on it; and that when silent and at rest she remained free from the oppression of which we have at first spoken, but when she began to do anything, or talked a little longer than usual, she was immediately seized with the anguish, which she used to describe by comparing it to that pressure and constriction which one feels from being squeezed by a great crowd of people. This oppression of the heart was also accompanied with slight faintness; the pulse was always feeble when at rest. The patient, as Albertini had predicted, was at length worn out by the disease. Everything in the thorax was found in a healthy condition, except that the pericardium was distended with nine ounces of water." Dr. Parry remarks on this case: "These symptoms are certainly at first view very similar to those of the angina pectoris. There is, however, this essential difference, that in the intervals between the paroxysms of the angina pectoris, when pure and uncomplicated, there is rarely any

serious affection of the pulse; and no sense of weight about the heart, or any other disorder; while in the simple dropsy of the pericardium, the pulse is, I believe, invariably changed, and the sense of weight or pressure on the heart, though always aggravated by exertions, constantly more or less exists, whether the patient move or be at rest." Of course, at the present day, the evidence furnished by physical signs would tend to discriminate the two forms of disease more clearly. But the summing up from the subjective phenomena is very instructive and may interest many readers.

Pericardial Adhesion.—One of the two common results of pericarditis is the adhesion of the two pericardial surfaces; a condition not incompatible with years of existence in many cases. The amount and extent of the adhesions vary; and with them the progress and prognosis of the case. Accordingly then:

The adhesions may be partial, and consist of one or more attachments drawn by the heart's movements into bands, resembling pleuritic adhesions.

There may be adhesions dividing the pericardial cavity into loculaments containing fluid or other contents.

The adhesions may be complete, and the pericardial cavity be thus obliterated.

The adhesions may be complete, and also contain the chalky debris of the fibrin of the effusion; or a sort of ring be formed of connective-tissue corpuscles in which lime salts became deposited—called by older writers ossification of the heart.

The adhesions may extend to and involve the costal pleura; and under these circumstances only are there any objective evidences of its existence. When, however, solid material remains behind unabsorbed, then there is an area of increased cardiac dulness; a condition closely simulated by a mediastinal tumor, or consolidation of the piece of lung overlapping the heart.

As to the changes wrought in the heart itself by adherent pericardium, they vary according to circumstances. Where the adhesions are partial then the heart is dragged upon, and this embarrassment leads to hypertrophy (according to the rules laid down in Chapter IV); where the nutrition is good, hypertrophy results; if the nutrition be not very active, then dilatation follows.

Where hypertrophy is established the incommoded and embarrassed heart is enabled to fulfil its functions for years with fair efficiency. In other cases, however, the muscular structure of the heart is involved; the muscular fibre is pale, and the subject of fatty degeneration. There are rarely wanting traces of myocarditis with its scar-like depressions. There is also, according to Rokitansky, in some cases a tendency for the inflamed pericardial surfaces to adhere along the track of the coronary vessels; and when this occurs there is early and extensive degeneration of the muscular structure of the heart from the blood supply being thus diminished. Of course it is clear that in these cases the state of the heart must be appraised from the evidences furnished by its condition, irrespective of any suspicion of adherent pericardium, if there be no physical signs of the adhesion.

The subjective symptoms of adherent pericardium are those of the condition of the heart with which it is associated. Where there is fair hypertrophy the patient may exist for years without any evidence of its presence. Thus Stokes had a case which went on seven years without any evidence of the existence of pericardial adhesion. Where degeneration of the heart is set up then there are evidences of heart failure, such as dyspnœa, readily aggravated on exertion, angina pectoris, venous congestion, visceral fulness and dropsy. Thus though there are no special subjective symptoms of pericardial adhesion, when these symptoms follow after extensive pericarditis, and gradually grow in severity, there is at least presumptive evidence that pericardial adhesion exists.

Corvisart indeed thought that adherent pericardium gave rise to flushings, and the sensation of dragging in the region of the heart, due to a downward traction of the heart and pericardium by the diaphragm during inspiration, and upward traction by the heart in systole (Hayden).

As to objective phenomena, they rarely exist where the costal pleura is not involved. Sibson says: "When the heart is, as usual, enlarged, being often affected with valvular disease, the adhesions may be short, fibrous, and binding; and the front of the organ may be fixed to the lower two-thirds of the sternum and the adjoining cartilages by pleuro-pericardial adhesion, so that the automatic and respiratory movements of the heart and

the inspiratory expansion of the lungs are restrained; thus the discovery of the adhesions during life may generally in such cases be made by a careful study of the physical signs; its diagnosis being the more certain and easy in proportion as the heart is more enlarged, and more firmly fixed to the anterior walls of the chest." There are, he says, according to Skoda, "the systolic indrawing of the lower sternum or intercostal spaces by the contraction of the adherent heart; and the diastolic shock or backstroke that immediately follows, given by the return elasticity of the chest-walls." According to Balfour, "depression of the præcordial region is much more rare than its elevation, and is the result of previous pericarditis, and the indication of adhesion of the visceral and parietal portions of the pericardium."

The retraction of the intercostal spaces on each systole is distinct in many cases, the heart dragging on the adhesions in systole—being shortened in systole and elongated again in diastole, and also turning on itself backwards and forwards in these movements. Hayden thinks "a fixed position of the apex point at or near the nipple may be regarded as eminently suggestive of adhesion." Hope thought pericardial adhesion gave a "jogging" motion to the heart; and Bouillaud says, "On sent à la main que la jeu du cœur est embarrassée." By percussion too there is furnished the evidence, and very important it is, that the area of percussion dulness is not altered, as it normally is, by expiration and inspiration. Friedereich holds there is diastolic collapse of the jugular veins. Hayden concludes: "If systolic pitting of an intercostal space at the point of apex-pulsation, and of the epigastrium, with absence of variation in the relative extent of pericardial dulness and vocal vibration during full inspiration exist, I should have no hesitation in concluding that both pericardial and pleuro-pericardial adhesion had taken place. Exaggerated systolic depression of the epigastrium, and of the lower end of the sternum, would indicate the twofold condition of general agglutination of the pericardium and hypertrophy of the heart." Pearson Irvine holds that the dragging of the heart on the pericardial bands leads to irregularity of action of the heart, especially in the early period of the adhesions.

The prognosis of pericardial adhesion is hopeless as to cure, and

bad as to duration of life. If it be followed by fair hypertrophy of the heart, as evidenced by a fair pulse, good general health, and fair physical power, it would be rash to hazard any opinion as to the probable duration of life. But when the evidences of heart failure from degeneration of the muscular walls manifest themselves after an attack of pericarditis, especially if it have been extensive, then no great prolongation of life is probable or can be looked for; as the causal conditions of the muscular degeneration are not to be removed by any remedial measures. The treatment of course is that of the muscular walls, which must be aided to hyperplasia by the measures detailed in Chapter XII. No one now would think of producing any effect upon the adhesions by the exhibition of mercury or iodide of potassium.

Hydropericardium.—Hydropericardium, or the effusion of fluid into the pericardial sac, is a non-inflammatory ailment, and is not to be confounded with the effusion following acute pericarditis. It is a serous effusion usually found with cardiac failure, and Bright's disease, as chronic conditions; and with scarlet fever as an acute condition. It is a mere effusion with a trace of albumen, without the lymph of inflammatory effusion, and when absorbed leaves no traces of solid material behind it. It has at various times been examined as to its constitution, and Wachsmuth gives the following as the results of his analysis:

Water from	95.37 to 97.34
Fixed material from	2.66 to 4.63
Albumen from	1.43 to 3.01
Other material from	1.23 to 1.64

It is often extensive in quantity, and is a source of distress to the patient; but is not commonly a cause of death. The pericardial sac is not large, and therefore great effusion is impossible; for any large effusion would compress the heart so much that filling of its chambers in diastole would be no longer possible. The amount is usually five or six ounces; but it may be much more. Quantities below an ounce cannot be regarded as pathological.

The effects of hydropericardium upon the heart is to embarrass its action: while the heart-fibre becomes pale and easily torn. This does not arise from degeneration of the muscular fibre, so

much as from infiltration with serum and impaired consistence. It may lead to diminution of the bulk of the heart from pressure, but this is not nearly so frequent as in the case of the effusion of pericarditis. The fat of the heart disappears in proportion to the duration of the effusion (Rokitansky).

The conditions under which hydropericardium is found are:

1. Scarlatina. Here it is part of the general effusion from serous surfaces which follows arrest of the action of the skin when desquamating, the renal tubules being choked with epithelial scales, forming the casts so characteristic of this post-scarlatinal condition. It comes on insidiously as a rule, though in other cases the effusion is comparatively acute; and is usually accompanied by pleuritic effusion, and at times by peritoneal effusion. It rapidly disappears on the re-establishment of the renal flow.

2. Bright's Disease. Hydropericardium is found in the general dropsy of advanced conditions of Bright's disease. It is as yet unknown whether under these circumstances the effused fluid contains urine salts or not. When occurring with Bright's disease, hydropericardium is more serious and more intractable than when it occurs after scarlatina; indeed it is a part of a more generally serious condition.

3. Chronic Heart Disease. It is found here as part of the general dropsy found in the later stages, where there is effusion from every serous surface. Its presence here adds to the embarrassment of the heart, and so aggravates the patient's condition. Whether the effusion comes from the external or parietal layer of the pericardium, or both, is a matter of speculative interest only. Niemeyer and Oppolzer hold that it is the result of engorgement of the right heart, and effusion from the coronary venules of the cardiac pericardium; but there is no reason to suppose that the external pericardium escapes the condition of the serous surfaces generally.

4. It may occur with a generally dropsical condition, as in acute nephritic dropsy; or in more chronic conditions of general dropsy, of which we know little pathologically.

5. Niemeyer is inclined to regard one form of hydropericardium as a species of compensatory effusion. He says: "We have already seen how a decrease in the size of the heart, by reducing

the pressure upon the pericardium from within, results in an increase in quantity of the fluid in the sac. The same thing takes place when the lungs become adherent to the pericardium, and are reduced in volume, either from atrophy, failure to regain their normal size, after absorption of a pleuritic effusion, or contraction from chronic pneumonia. This form of hydropericardium is analogous to the increase in the amount of cerebro-spinal fluid which takes place in atrophy of the brain, and as the latter is called hydrocephalus *ex vacuo*, so hydropericardium *ex vacuo* would be a suitable name for the former."

6. It has been found along with miliary tubercle of the pericardium in acute tuberculosis. Here its interest is purely pathological. The symptoms of hydropericardium are those of cardiac embarrassment, as given at page 323; of course there will be present the symptoms of the general condition of which it is but a part. The symptoms produced by it may be slight; but when the effusion is large, the embarrassment of the heart is great. There is dyspnœa, and the patient is compelled to assume the upright posture, to be propped up in bed, or even to sit in a chair. The effusion impedes the flow in the venæ cavæ, and aggravates the cyanosis and the dropsy, according to Niemeyer. The objective symptoms are diminished apex beat, with somewhat of bulging of the chest-wall in young subjects; there is also diminution of the intercostal depressions over the cardiac area. The impulse of the heart is feeble, and "may be very perfectly undulatory" (Walshe). On percussion, there is an increase of area of cardiac dulness, increased laterally on lying down, and resembling to some extent a triangle lying on one of its sides. Percussion, however, may be interfered with by the condition of the lungs. On auscultation the heart-sounds are heard clear and distinct, but not loud; there is no friction-sound at any time. The pulse is feeble, but not necessarily irregular.

As to prognosis, this depends upon the associated general condition. In scarlatina, it is usually good; in kidney disease it is certainly serious, though much less so in acute nephritic dropsy than in chronic renal disease. In chronic heart disease it is usually developed at the close of the case, and is commonly a precursor, if not the cause of death. In chronic lung disease, or

heart atrophy, the hydropericardium *ex vacuo* is not likely to disappear; in the dropsical dyscrasis it will depend upon the general condition. Water at or round the heart are common expressions about the state, and usually the last state, of dropsical patients, and its presence is regarded as very ominous. In the North of England, it is not uncommon to hear the expression, "He will live as long as his heart can swim," in cases of dropsy.

The treatment of hydropericardium is that, of course, of the condition on which it depends. In scarlatinal dropsy hydragogue cathartics, the warm bath, and diuretics are the measures to be employed; and the treatment is the same in acute renal dropsy. In post-scarlatinal dropsy, Dickenson advocates the washing out of the renal tubules by copious draughts of water, and the administration of digitalis to raise the arterial tension and increase the flow in the glomeruli of the Malpighian bodies; by these means the casts may be washed down the tubules. In chronic kidney disease, free purgation and diaphoresis are to be attained by appropriate means. Potash, colchicum, and buchu or juniper, may be given after acute kidney congestion is past. In chronic heart disease the dose of digitalis may be increased; but if the treatment adopted previous to the effusion into the pericardial sac has been at all active and judicious, little can be expected therefrom. In each case the treatment must be guided by the exigencies of the patient.

Hæmopericardium.—This may occur from some injury to the pericardium, and, if not excessive, recovery may take place. (See p. 247, Chapter X.) When due to rupture of the heart, death, sooner or later, is the result; but it may not occur for some days.

Pneumopericardium and Pyopericardium.—These conditions are usually found together, though one may precede the other. The combined condition may arise from suppuration within the pericardium, with the pus opening outwardly as an abscess; or an abscess may open into the pericardium, and gas be evolved from chemical changes in the pus. If the abscess be in the lung, air may find its way into the pericardium. The evidence of air in the pericardial sac is a clear percussion-note; indeed, the cardiac area may be resonant, or even tympanitic on percussion. The

tinkling of succussion may be heard. There is also general collapse.

The pericardium is the seat of tubercle and cancer at times, while hydatid cysts are not unknown.

For the relief of the above conditions Bamberger and Friedereich recommend the use of an exploring trocar, and the subsequent injection of chlorine water or iodine. Irrigation with an antiseptic solution might be tried as a forlorn hope.

CHAPTER XIV.

THE NEUROSAL AFFECTIONS OF THE HEART—NEUROSAL PALPITATION—GRAVES'S DISEASE—CHOREIC PALPITATION—INTERMITTENCY—IRRITABLE HEART.

Whatever difference of opinion may exist as to the increasing frequency of organic disease of the heart, unanimity obtains as to the growing prevalence of nervous affections. In the last of a series of lectures delivered at the West London Hospital, in the spring of 1878, and published in the "Medical Times and Gazette" in the autumn of that year, the broad question is put—"Are the nervous disorders of the heart on the increase at the present time?" To this an affirmative answer is returned. Dr. Crichton Browne asserts that the battle of life is now no longer fought with thews and sinews as of yore; the brunt of it now is borne by the nervous system; and, as a natural consequence, disease and disturbance of the nervous system are increasingly frequent. That in the present hurrying stage of existence, amidst excitement and worry, the heart should be disturbed in its action is only what might *à priori* be expected. Dr. Da Costa found in the American Civil War a cardiac malady before undescribed, which he denominated the "Irritable Heart," of which a description will be given in a later section of this chapter.

Such being the case, it becomes necessary to present these affections before the reader, as clearly and lucidly as the present state of our knowledge will permit.

Neurosal Palpitation.—Palpitation of neurosal origin is more frequent than palpitation, the evidence of muscular inability. It has been stated by several authorities, that when a patient enters the physician's consulting-room complaining of the heart primarily, the case is one of neurosal disorder. The patient with organic disease of the heart, complains of one of the consequences thereof—dyspnœa on exertion, or an intermittent pulse.

Such is my own experience. Palpitation is largely a woman's malady.

In Chapter III, palpitation is briefly reviewed, and the different forms of it contrasted with each other; but here it will be necessary to treat the subject at greater length.

In Chapter V, palpitation, as the result of muscular failure, is described; and it is stated that such palpitation is associated with evidences of heart failure, as dilatation with or without valvular disease. It is provoked by effort; or by a full stomach or colon pressing the diaphragm upwards against the heart, especially when the right ventricle is dilated. It may occur at night after effort in the preceding day, or after a liberal supper. But it rarely manifests itself during general quiet. It indicates that the muscular walls of the heart are not quite equal to the demand upon them, and if found with hypertrophy, tells that it is insufficient—not, as was once thought, that it is excessive. Then the relations of palpitation to arteriole spasm will be described in the next chapter, in relation to the changes found in the vascular system in chronic renal disease. At present, the subject of neurosal palpitation is alone being considered.

In order to comprehend nervous palpitation, the subject must be divided into (1) primary, and (2) reflex palpitation. In the introductory chapter, the innervation of the heart is described, and it is shown that, in addition to the primitive heart ganglia, there are many nervous communications with the sympathetic, on the one hand, and the cerebro-spinal nervous system on the other. By these communications the reflex disturbances of the heart are brought about. As our knowledge of this subject widens, probably the cases which we are compelled to regard as primary neuroses of the heart, at the present, will be found to be reflex in many instances. In the absence of any apparent source of irritation to cause reflex palpitation, we assume a neurosis of the cardiac ganglia. Such, probably, is the case in the palpitation of emotion; though here the vasomotor system may be involved, and palpitation be induced by arteriole contraction, leading to a rise in the arterial tension, and, consequently, of the pressure within the heart. In some women palpitation is very troublesome to them, being induced by comparatively slight causes. A

knock at the door, any sharp sound, the receipt of an unexpected letter, are sufficient to excite palpitation. Crossing a street becomes a gigantic task to such women; while the moderate marital attentions of their husbands can no longer be endured, in consequence of the sequential palpitation so excited. Any change in the nervous system provokes disturbance in the heart; and one of the most annoying and trying of them all is the severe palpitation set up just as sleep comes on. In other cases it wakens the patient out of sleep in the dead of night, and alarms her seriously. This alarm is aggravated by the intermissions which occur amidst the palpitation. It is common to hear the patient complain: "It is not the palpitation which frightens me so, for then I know the heart is going; it is the sudden stoppage which alarms me!" Any one can readily conceive how alarming such experiences are to a nervous woman alone in the dead of night. In some cases the heart beats in an excited manner persistently, with attacks of violent palpitation at intervals. Nor is this found in nervous excitable women only; I have met with it in women possessed of an unwonted amount of self-control. Nor, again, is any muscular weakness of the heart necessary for such palpitation. Hearts of this description might be described as "badly-behaved," or excitable hearts—*i. e.*, excited action is set up by causes too slight to evoke palpitation in other persons, and interfere much with their owner's peace of mind and power of labor.

In an attack of hysteria there is almost always palpitation of the heart, but then there is also a corded condition of the arteries, a high blood-pressure, and a rapid secretion of urine. During the attack the heart beats violently, as if it would shatter off the anterior wall of the chest; the hands and feet are cold, and the countenance is pallid; while the arteries are tense and incompressible. Here are all the evidences of vasomotor spasm, in which the heart is involved. After the attack is over the patient passes an enormous quantity of limpid urine of very low specific gravity. Such palpitation is scarcely fairly to be denominated primary palpitation of the heart; yet still less can it be classed under the next division.

Reflex palpitation of the heart is a very common occurrence, and as our power to recognize it increases, so does its apparent

frequence. The disturbance in the heart is excited by irritation set up in other viscera, and especially those of the pelvis. Thus the palpitation, excited and maintained by a prolapsed uterus, is well known ; and its disappearance on the restoration of the womb to its normal situation is conclusive evidence as to the association of the two. Ovarian excitement is a very common cause of palpitation. In an article entitled "Ovarian Dyspepsia," in the January number of the "American Obstetric Journal," 1878, the writer describes how ovarian irritation sets up far-away disturbances. We know that if a series of ivory balls be suspended in a row and a blow be given to the end one, it is the terminal one at the other end which flies from its place. So with nerve currents ; the disturbance is not felt at the seat of the primal irritation, but at the terminal point where the nerve current runs out or terminates. Thus we find in women ovarian excitement which not only provokes and maintains uterine disturbance and affects the catamenial flow, but which sets up also gastric disturbance, in the form of indigestion, cardiac disturbance as palpitation, respiratory disturbance as cough (like the cough of pregnancy, known in Scotland as "a cradle cough") ; and usually pain under the heart, so much complained of by women. This last is a true neuralgia excited by the ovarian disturbance. It would appear that waves of nerve perturbation may arise in an ovary, may travel along a series of nerve fibrils, and terminate ultimately in the peripheral endings of the intercostal nerves, where they are felt as gusts of neuralgic pain. The nerves so affected are the left sixth and seventh intercostals, rarely the right. The waves of perturbation set up in the ovaries may also run into the cardiac ganglia and cause paroxysms of palpitation. The patient, so afflicted, commonly associates the pain in the side with the palpitation, and supposes her heart to be diseased. But reflex palpitation is not confined to primary disturbance in the reproductive organs, it may be causally associated with the stomach, the liver, and possibly other viscera.

When palpitation is so excited it is very intractable, and its cure is unattainable unless the seat of primal disturbance be borne in mind. Digitalis and belladonna are of no avail or very little ; digitalis often making the condition worse. Bromide of potas-

sium is the great agent to be relied upon, as it acts as a sedative to every part of the nervous system involved; alike the peripheral nerve-endings in the ovaries; the nerve tracts along which the nerve currents pass; and the cardiac ganglia in which they terminate. To keep the pelvis free from fecal load is very desirable; and small blisters over the ovary are of service.

The sex in which such palpitation is usually found is of course the female sex. The emotional nervous system of woman is less controlled, or inhibited by the higher centres in the cerebral hemispheres, than is that of man; as well as being more sensitive. But it is not confined to women. Recently a male patient consulted me. He was a man of active sexual inclinations whose wife had died suddenly. Here repression of the sexual appetite gave rise to violent paroxysms of palpitation, at once relieved by intercourse. Thus we see that continence may give rise to palpitation, as well as indulgence of the sexual passion. In consequence of its relations to the reproductive organs the heart is liable to attacks of palpitation at puberty; and still more at the menopause, especially if there be then much recrudescence of the sexual passion. In one case, in a young gentleman, the cause of the palpitation was long sought for in vain; at last rectal irritation was discovered. On appropriate treatment directed to the cause of the rectal irritation, the reflex palpitation passed away completely.

Botkin, of St. Petersburg ("Maladies du Cœur"), speaks of intercurrent irritation setting up palpitation of the heart; and instances a floating kidney. There is a strong probability of prostatic disease being a source of palpitation; and one such case has come under my notice; but as the patient did not repeat his visit, I cannot say whether he received any benefit from the treatment suggested or not. He had travelled far and wide, and tried many doctors and numerous springs without satisfactory results before seeing me; and the probability is that he is still engaged in a futile search after relief. The subject of reflex palpitation is one on which as yet very little is known; and it needs thorough investigation. The patient with prostatic disease was a most excitable being; and it is probable that reflex excitement of the heart in man is more easily produced where the nervous system gener-

ally is unstable, or where it approaches in type the feminine nervous system, than in those constituted otherwise.

Where neurosal palpitation of the heart is long continued trophic changes may be set up. We saw in Chapter V how motor discharges of nerve force not only set up muscular action, but dilated the bloodvessels of the muscle made functionally active. Hope recognized a nervous form of hypertrophy of the heart (p. 114); and how such a form of heart-change is brought about is now explicable enough. Where such hypertrophy exists the heart's action is persistingly excited, and there is a strong apex beat. But the diagnosis is not easy to make, or one to be very certain about in many cases; as there are many sources of fallacy.

The diagnosis of nervous palpitation is made by the consideration of the general surroundings of the palpitation, rather than from any physical signs. The negative indications distinguishing it from other forms of palpitation, are the age and sex of the patient, and the absence of murmur or other indication of valvular disease; the character of the pulse, which is small and quiet, and bears no relation to the excited action of the heart; and the absence of arterial changes. The positive indications are its not being affected by effort, its presence during quiescence, a neurosal diathesis, and the evidence of sources of reflex disturbance elsewhere.

The prognosis of nervous palpitation is good as to life, but very unsatisfactory as to cure. Where it is due to a neurosal diathesis, it may be palliated, but the tendency to return is strong. When associated with ovarian congestion it is fairly amenable to treatment; when due to prolapse of the womb it may be relieved at once, and permanently, by replacing the womb in its normal situation. Frequently it is due to tea, and it is quickly relieved when cocoa is substituted for the tea. It would be interesting to know if palpitation is produced by drinking coffee to excess; but, so far as I know, no observations have been made on the subject. Something more will be said later on as to the effect of toxic agents upon the heart. Palpitation which obtrudes itself forcibly upon the patient's attention is usually nervous in character. The palpitation of organic disease is rarely violent, and, when it is so, it is usually associated with effort, and a heart in the later stages of

failing hypertrophy. Occasionally an elderly patient rather likes to show the medical attendant how easily violent palpitation is excited by a few quick steps across the floor. In advanced conditions of structural disease of the heart-walls, palpitation is rather a good indication, showing that the heart can still palpitate; and in chronic irregularity palpitation on effort is not nearly so serious as syncope, or intermittency so produced.

True nervous palpitation may indicate nervous exhaustion; and its recurrence at shorter intervals, or in greater force, or for more prolonged periods, is not a symptom to be undervalued. It may indicate mental strain, and tell that the nervous system is tried from its psychical and not its physical side. As seen in the case above it may be the result of continence in the male, and in celibate females may indicate that the nervous system is suffering from the enforced involuntary continence they practice. In other cases it is set up by intercourse, which may be in no way excessive, and is the cause of much suffering to many married women. In one case known to me the marital relations had been suspended for seven years in consequence of the cardiac disturbance occasioned thereby. After a course of quinine and hydrobromic acid, continued for some months, the patient became perfectly well and resumed her relations with her husband without discomfort. The prognostic import of neurosal palpitation is related not so much to itself, as with the condition to which it belongs, or in which it takes its origin. Peter Frank is a well-known instance of palpitation induced by arduous study of heart disease; and the action of the heart can be influenced by attention to it—as most medical students know for themselves. Where nervous palpitation alarms the patient it is more likely to perpetuate itself.

The treatment of nervous palpitation divides itself into the treatment of the attack, and the treatment of the interval. During the attack the patient should lie down if possible, and be allowed perfect quiet; all emotion on the part of those around being suppressed as far as possible. A little alcohol may be indicated, or sal volatile, or smelling salts, especially in attacks of psychical origin. The late Professor Laycock thought that certain animal odors, as musk for instance, were valuable in emotional disturbances of erotic origin. If the attack be of the nature of hysteria,

then C. J. Hare's plan of holding the nose until a long and deep inspiration is produced, is preferable to dousing the patient with a pitcher-full of water. Where the palpitation is set up by sudden effort a diffusible stimulant with a little belladonna or digitalis is indicated.

The treatment of nervous palpitation in the interval is a somewhat difficult subject, as it involves the condition on which it depends. When primary, it is well to try quinine with hydrobromic acid, a combination which has several times given me very satisfactory results. At other times when associated with anæmia, tonics and chalybeates, as Easton's syrup, are indicated. When reflex, the cause of the disturbance must be carefully sought for, and removed if possible. Then, if ovarian, it is well to place a little blister over the affected organ, especially at the catamenial periods, and to keep the bowels open by sulphate of magnesia, so as to do away with any load in them, which always aggravates any disturbance in the pelvic viscera. Camphor mixture forms an excellent vehicle for these not very palatable remedial agents; and this combination should be continued over two or three mensual cycles. At the same time the discharges which are the outcomes of uterine turgescence must be attended to, and the use of astringent injections enforced. By such measures combined, and a belladonna plaster over the heart, great relief, and not unfrequently a cure may be attained; but it is rare to find any great result attained quickly; the treatment requires time and patience.

As to the psychical relations of nervous palpitation, they are too important to be overlooked. Not uncommonly it is associated with a neurotic temperament which is readily perturbed by slight exciting causes. But in other cases the life led has much to do with it. Indoor occupations foster it, and fresh air and exercise are essential to successful treatment.

The sensational character of modern fiction is not without its effect upon the nervous system of its votaries; and the enthralling plot which the confirmed novel reader demands is allied to the demand for brandy in the toper—less potent stimulants are insufficient to rouse the jaded nervous system. Still worse, it is to be feared, is the pruriency which is making its way into fiction, and especially the works of some authoresses. Poisoned by such moral

filth the nervous system of the reader becomes still further unstable, and still less under the control of the higher centres. In such cases, removal from the neighborhood of a circulating library is a necessary step; and exercise, the occupation of the mind with other subjects, are desirable. How far an involuntary celibacy is a factor in the production of cardiac disturbance amongst other neuroses, is a subject somewhat difficult to discuss. The mental processes of women are skilfully concealed by them, and there are natural and great difficulties in the way of such inquiry as alone could light up the subject. With matrimony as the sole future for most women, a spinster's thoughts may often turn in an erotic direction; nor must we be uncharitable and condemn her for this. We can find allowances for soldiers where the military regulations do not permit of their marrying! Why can we not then be equally charitable to those of the weaker sex, who are also compelled by the stern pressure of their surroundings to remain single, and yet who have even less to occupy their attention in other directions? That celibacy is the voluntary choice of those sexless beings, who chance to be cast in a feminine mould, may not be doubted; it is also at other times the choice of other women, who seek in religious exercises and devotion to subordinate their passions and their appetites, but who are not at all times and always perfectly successful; the flesh asserting itself in some morbid neurosal manifestation, highly interesting from a psychological point of view. The impulses or promptings of the affectional nature may become stimulated by a life of constrained celibacy, and the ovarian excitement and the nervous centres may act and react upon each other, until occasional promptings may develop into a persistent furor, as the wards of our lunatic asylums only too amply testify.

The experiences of St. Catharine in her convent cell are not unique, and the erotic dream is the safety-valve of many a nervous system strained to the utmost point of tension among our single sisters.

The Germans have the expression *ein Liebes Traum*, and the French *le rêve voluptueux* for this form of dream with orgasm. There is also a coarse English expression which may not be quoted here. "The sexual appetite appears only with the development

of the generative glands. Its appearance induces considerable perturbation of the other organic functions, and expresses itself subjectively at first, chiefly in the form of emotional excitability, or in obscure longings, morbid desires, or hysterical outbreaks. Long before the link between a definite sensation and a definite action for its realization has been established in consciousness, the generative glands may gratify themselves reflexly during sleep, the period *par excellence* of reflex excitability" (Ferrier).

Many cases of nervous palpitation are cured when the cares of maternity are added to the caresses of a husband; when the emotions get settled down into healthy channels, and the natural aspirations attain their physiological gratification. In speaking of the treatment of hysteria Russell Reynolds says wisely, "It does not consist in the administration of nauseous gums, but in proper mental, social, and moral management." Be it ever remembered that in the sane as well as in the insane, the mental condition is always aggravated by and rendered less tractable, when there exists a physical factor present; and in those patients where the *fons et origo malis* may seem to be a psychical condition it is well to examine the case thoroughly; and in many instances it will be found that some physical condition is present which may be treated therapeutically; and so the patient be materially aided in her attempts at self-cure. Mental treatment and rational therapeutics may be made auxiliary to each other in many cases with advantage; and a well-selected mixture may be very useful and something more than ancillary to a course of devotional self-subordination.

Graves's Disease: Exophthalmic Goitre.—This is a neurosal affection in which the heart is involved, and is a complex malady which has attracted much attention among intellects of a high order. The names of Basedow, Graves, Stokes, Begbie, Aran, Trousseau, Remak, Von Græfe, Oppolzer, Traube, Niemeyer, Virchow, Von Recklinghausen, Charcot, and of others, are connected with the study of this malady; and yet we have not progressed beyond certain clinical data, and are as far from any real knowledge of the nature of the malady as ever. Yet this malady has distinct characteristics. There is prominence of the eyeballs; enlargement of the thyroid gland; and tumultuous action of the heart. In the

first edition of this work I ventured to add a fourth characteristic, viz., a decidedly emotional temperament; and further experience encourages me to think the fourth as certainly a part of the disease as any of the other characteristics. It may be well to consider each symptom separately, as this malady has no distinct "pathological anatomy." The scalpel and the microscope have done nothing yet to unravel the mystery of this curious affection. Clinical observation has suggested that it is a neurosis of the sympathetic (Trousseau); while Niemeyer thinks the vasomotor nerves of the coronary circulation are specially implicated. Under these circumstances it may be well to review each characteristic separately.

The heart symptoms consist of decided tumultuous action, extreme rapidity of pulse in many cases, amounting even to 160 or 200 beats per minute; the heart seeming as if thrown bodily against the thoracic parietes, while its impulse can be felt over a large area. There is usually some enlargement of the heart, and increased dulness; and the heart resembles in every respect a dilated and hypertrophied heart under excitement. There may also exist valvular disease, but, if so, it is a mere coincidence, and no part of the disease. A systolic murmur may be heard chiefly at the apex, and even a thrill felt at times, but this probably occurs from disordered action of the musculi papillares. As well as the cardiac disturbance there is also abdominal pulsation, and the carotids are full, and may even give rise to a thrill.

The thyroid gland is enlarged, and it may vary in bulk from time to time. It has not the firm feel of bronchocele, but is elastic to the touch and pulsates. The enlargement is due to dilatation of the branches of the thyroid arteries, rather than to a development of connective tissue, or gland structure. Under excitement the thyroid is enlarged and the sensation of pulsation increased. There may be a thrill, or even a murmur; or the lobes may be unequally enlarged.

The eyes are prominent, and in well-marked cases the prominence is sufficient to establish the diagnosis. The expression in the eyes varies; where there is little prominence the eyes are merely full, giving an interesting expression to the features; but when the prominence is very marked the eye has a wild or scared

look, like that of a hunted animal. In some cases the protrusion of the eyeballs is such that the eyelids are unequal to closing over them, even in sleep. This together with the tension leads to various changes in the cornea. A catarrhal condition may be established; or there may be ulceration; or the cornea may be infiltrated with pus-corpuscles. The upper eyelid becomes swollen and the enlarged blue veins in it become conspicuous. Von Græfe regarded a retraction of the upper eyelid with a downward cast in the eye itself as pathognomonic of this affection. There is no alteration in the pupil, nor changes revealed to ophthalmoscopic examination.

The temperament is emotional, susceptible, and sensitive; and women with Graves's disease are easily upset by trifles, and attach importance to matters which are regarded as too trifling for notice by others. Consequently they make many disturbances, and if married have quarrels with their husbands; if unmarried the favorite brother is the common subject of the emotional *émeute*. Many, indeed most of such patients, readily admit this tendency to emotional outbursts, which are without any real foundation or cause, but which are as uncontrollable as they are involuntary. These patients very commonly possess a refined temperament, and such ebullitions distress them much. There is a neatness of attire and a carriage about most women with Graves's disease which distinguish them at once from the bulk of hospital out-patients. Along with this emotional temperament there are usually catamenial disturbances, the flow being either scanty or profuse; while there is frequently leucorrhœa present.

Women are much more frequently the subjects of Graves's disease than men, as might be expected from their emotional temperament. Von Græfe found only one man in every seven patients affected with this malady. The period of life at which Graves's disease is found is that of the reproductive period, from puberty to the menopause; it is rarely seen before or after this period. In man adult life is also the time during which this malady is manifested.

Changes in the calibre of the arteries of various parts constitute the pathological change of this condition. Thus the small arteries behind the eyeballs enlarge, and this, together with the develop-

ment of fat-cells, causes the eyeballs to become abnormally prominent. The development of fat-cells may cause a permanent condition of prominence; but the variations in the amount of prominence must depend upon the variations in the calibre of the vessels. The enlargement of the thyroid, which also varies from time to time, is likewise due to changes in the calibre of the thyroid vessels. The disturbance in the heart may be due to disorder of the vasomotor nerves of the coronary circulation, leading to dilatation of the vessels and so of altered nutrition, by which the heart may become enlarged, and its action exalted. Or to some extent the heart-changes may be due to variations in the calibre of various vessels by which the blood current is obstructed; for dilatations in a tube as well as contractions in it interfere with the flow of fluid within it.

It has been thought that in some cases there is an enlargement and redness of the inferior cervical ganglia with sparseness and diminution in size of the ganglion cells, and a development of fat therewith; but any pathological change sufficient to account for the symptoms has not yet been observed, and has still to be discovered. Pressure on the structures connected with the eyeballs, may lead to degeneration of the muscles, or to atrophy of the lachrymal gland. Cerebral hæmorrhage has been found.

The affection comes on from various disturbing causes, physical or mental, in a manner not yet explicable; as, for instance, the wife of an American physician consulted me recently with this malady well marked; it came on quite suddenly as the result of the shock of her father's sudden death; up to that period she had had no indications of it whatever. The favorite subjects of it are fair women with light-colored hair, and blue or hazel eyes, who are of the diathesis termed nervous by Laycock, and who are highly sensitive and emotional.

The duration may extend from a few weeks to years. The condition varies, at times slight cases get almost well, and then are worse again. The aggravated condition found at times in severer cases passes away after a variable period. As to the prognosis it is very unsatisfactory. Death is a rare termination, and the affection, though intractable, is rarely so serious as to threaten life. Of fifty-six cases, Von Dusch had fourteen complete re-

coveries, and great improvement in twenty-six, while there was no improvement in four, and seven died, the result being unknown in five cases. Von Græfe had twelve per cent. of deaths, twenty per cent. of recoveries, thirty per cent. of great improvement, and thirty-eight per cent. unknown.

The diagnosis is easily made by the eye in well-marked cases. There are the prominent eyeballs, the enlarged thyroid, and tumultuous action of the heart, with emotional perturbation readily induced. The first two are sufficient evidence when seen together. The case is, however, at times obscure, because the prominent eyeball and thyroid enlargement may be wanting, and there is only the cardiac factor present; but here the temperament is very significant. In addition, Trousseau thinks we have an ancillary piece of evidence in "*tache cérébrale;*" that is, the production of bright red flecks or spots on the skin of the head by slight irritation by the nail. This is a transitory and localized vasomotor paralysis of the minute vessels of the skin immediately under the surface irritated. Trousseau thought these "*taches cérébrales*" pathognomonic of the "neurosis of the sympathetic;" but they are not quite that, though diagnostically valuable.

The treatment is unfortunately in many cases very unsatisfactory. Digitalis does not usually yield the desired results which might fairly *a priori* be expected from its action upon the vasomotor nerves, though it is worth trying. Belladonna is a remedy of repute in this affection. Ergot might be tried. Iron is useful where there is also anæmia, though Trousseau is not in favor of it generally. Preparations of iodine have been praised, and Oppolzer strongly advocated the combination of iron with iodine, as in Blancard's pills, for instance. Remak advocated the use of the constant current, which may be tried without fear. Where there is much cerebral excitement bromide of potassium is indicated.

Beyond the medicinal treatment there is the moral management of the case, and the patient must be encouraged to recover her self-control, and to resist the emotional disturbances as far as is possible. Gentle kindly behavior towards these patients also does them good. Everything physical and psychical likely to upset or excite such patients must be studiously avoided. All catamenial troubles must be remedied as far as possible, and leu-

corrhœal losses attended to. If the diathesis or temperament be beyond our reach, we may still do much to add to the patient's comfort by attending to the various little matters over which we can exercise control.

Choreic Palpitation.—In chorea the heart commonly beats violently as well as tumultuously, resembling the condition common in Graves's disease. As to the cause of this, nothing is yet known. The whole heart seems thrown violently against the chest-walls, which vibrate from the shock. There is often a murmur heard, of no precise locality, but systolic in time.. It is supposed to be due to irregular action in the musculi papillares. The cardiac complications usually clear up with the general improvement which comes in time. The best remedial agent is iron, with bromide of potassium, or with arsenic; in some cases zinc or copper are useful; while belladonna suits other cases, and in the form of a plaster is beneficial in all cases. Digitalis does little good here.

Intermittency.—Intermittency of the heart's action is a phenomenon about which more knowledge than as yet obtains is very desirable. The subject is reviewed in Chapter III, but something more may be stated here. Below are given three diagrammatic illustrations of the different forms of intermittency. They are not pretended to be strictly accurate. The white lines are the heart-strokes as felt in the radial pulse. They may be imitated by tapping the finger on a table or other resonant body. The first is that of simple intermittency, amidst steady rhythmic

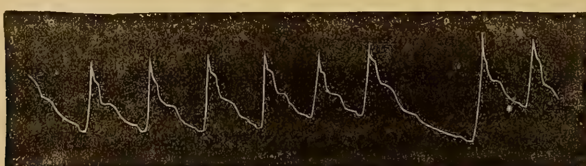
strokes there comes a distinct pause, after which follows a longer, stronger ventricular stroke. The second is the form found with failure of the muscular walls of the heart, and commonly with

dilatation. Here it will be seen that several normal beats are followed by two short sharp beats, then a long pause, and a long

strong stroke; after which the normal strokes are repeated for some time, often much longer than the limit of the scheme will per-

mit of its illustrating; and then come the two or three sharp short strokes, followed by the long pause and the strong systole. The third illustrates the form of intermittency of the pulse which is due to the ventricular stroke being too feeble always to reach the radial pulse. Here there is not a long ventricular stroke after a pause, because the pause is not due to a ventricular halt. The heart will be found beating rhythmically while intermittency is pronounced in the radial pulse. The ventricular contraction may reach the radial pulse as a feeble pulsation, as seen in the left-hand portion of the scheme; or one is lost altogether after a feeble contraction fairly perceptible, as seen in the later portion, or right hand of the scheme. There may be a long pause, but there is no unusually long strong ventricular contraction. By contrasting these three forms the student will readily learn the peculiarities of each.

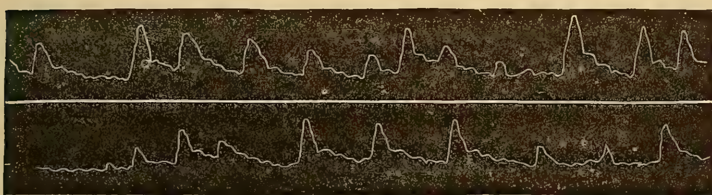
In order to further illustrate this subject, which is one of great practical importance, some sphygmographic tracings will be given. They were taken by Dr. Pond, of Rutland, Vermont, U. S. A., with his new Phonographic Sphygmograph, and manifest the different forms of intermittency very clearly. The first gives the mere nervous halt very clearly. It may occur frequently, as every



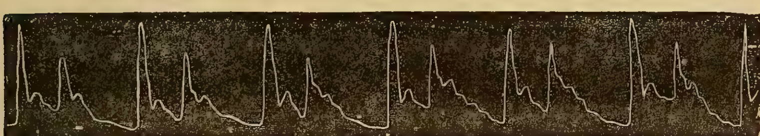
fifth or seventh stroke; or at longer intervals, as every twenty-second or forty-first. This form of intermittency is of no prognostic significance. It may be found with perfectly healthy circulatory organs; or it may be, and commonly is, found in

elderly men with enlarged hearts and atheromatous arteries, where it is also without any special significance, and will continue for years without alteration. As the condition worsens the intermittency changes its character. This is the form of simple intermittency so well described by B. W. Richardson, F.R.S., as occurring independently of organic disease.

Then comes the tracing indicative of serious organic disease, of which dilatation is always a part. Here it will be seen there is irregularity blended with intermittency, and some feeble beats precede the long interval and succeeding forcible ventricular stroke. This is the form of intermittency which is significant of muscular

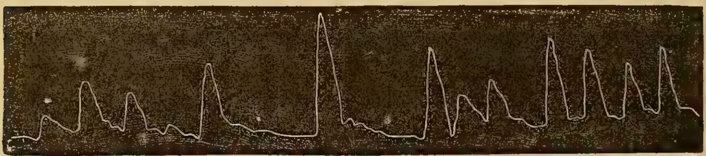


failure. This double tracing is taken from a very pronounced case. Here the action of digitalis will be found to be beneficial and to steady the pulse if given in sufficient doses. Then comes the intermittency of an incomplete ventricular contraction. Here the



normal stroke and the imperfect stroke are given as occurring alternately, and though an admirable tracing for Dr. Pond's purpose is scarcely so well fitted for mine; it illustrates the form of pulse well, but the irregularity is too regular for the majority of cases. Still it contrasts well with the preceding tracings. Such intermittency, complete, or, as here, incomplete, may be taken as indicating fatty degeneration of the walls of the heart.

Finally comes the intermittency of approaching death, where the tops of the tracings are rounded, an indication of sinister omen in all cases of failing pulse. The student should study these different forms of intermittency very carefully, as the information furnished thereby is of the greatest value at the bedside.



Irritable Heart.—Under this heading I think it will be well to include conditions spoken of in my first edition as Muscular Subparalysis and Hyperæsthesia of the Heart, as really the maladies so described are but forms of the irritable heart. This term —the irritable heart—was first used by Da Costa, of Philadelphia, in describing a form of heart mischief, hitherto unrecognized, which was frequently seen in the American Civil War.

The following abridgment of Dr. Da Costa's description is in his words:

"The general clinical history of many of these cases was this: A man who had been for months or longer in active service, would be seized with diarrhœa, annoying, but not severe enough to keep him out of the field; or attacked with diarrhœa or fever, he rejoined, after a short stay in hospital, his command, and again underwent the fatigues of a soldier's life. He soon found he could not bear them as formerly; he got out of breath, could not keep up with his comrades, was annoyed with dizziness and palpitation, and with pain in the chest; his accoutrements oppressed him, and all this though he appeared well and healthy. Seeking advice from the surgeon of the regiment it was decided that he was unfit for duty, and he was sent to a hospital, where his persistently quick-acting heart confirmed his story, though he looked like a man in sound condition.

"Any digestive disturbances which might have existed gradually passed away, but the irregularity in the heart remained, and

only very slowly did the excited organ return to its natural condition, or it failed to do so, notwithstanding the use of remedies which control the circulation; thus the case might go on for a long time, and the patient, after being the round of the hospitals, would be discharged, or, as unfit for active duty, placed in the Invalid Corps."

Thus it will be seen that there is actual impairment in the power of the heart, though the affection is a nervous one; and a further experience enables me now to say that the irritable heart in many respects simulates the enfeebled heart of organic disease, especially the dilated heart. But the absence of the physical signs of a dilated heart, and the character of the pulse establish the diagnosis, *i. e.*, taken together with the patient's history. The pulse of each form is unrhythmical to some extent; but in dilatation the irregularity is more marked. Some of Da Costa's cases seemed almost entirely due to exhaustion of the sympathetic, especially of the cardiac ganglia, and these yielded readily to treatment and rest; while others assumed more the characteristics of Graves's disease, and were scarcely, if at all, benefited by treatment. The tendency was towards the development of hypertrophy, and in some cases this became marked, but usually it was trifling in amount. In one case, a fairly typical one, death from a strangulated hernia furnished an opportunity of examining the heart.

"The pericardium was healthy; the heart, before being opened, appeared to be of normal size; at its upper portion was a moderate amount of fat; the valves were all healthy; a small clot was entwined in the mitral valve; the auricles were of normal size, and so were the cavities of the ventricles; the muscular structure of these was firm, and the cut surface glistening. While, as already stated, the cavity of the left ventricle did not appear increased, there was a great disproportion between its walls and those of the right side; these measured less than one-fourth of an inch in some parts, a fraction over one-fourth at the thickest portions; whereas the walls of the left ventricle were nearly seven-eighths of an inch at the thickest part, and varied from a little over one-half to three-fourths of an inch at others. Microscopically examined, the fibres were healthy; some fibres seemed indistinct, but there was neither fatty nor granular degeneration. The nervous filaments

of the heart, as far as they were traced out, appeared healthy, but no minute dissection of the heart was made." The physical signs in life were "impulse somewhat extended, but not decidedly abrupt, and of some force; the second sound is very distinct."

As to the causes, Da Costa says: "In no part of this inquiry is it more difficult to arrive at fixed conclusions, for many causes seem at times to have been combined, and it is scarcely possible, even by the most rigorous analysis, to fix especially upon one." Of 200 selected and well-marked cases, the analysis stands thus:

Fevers,	34 or 17 per cent.
Diarrhœa,	61 or 30.5 per cent.
Hard field service, particularly Excessive marching,	69 or 38.5 per cent.
Wounds, injuries, rheumatism, scurvy, Ordinary duties of soldier life and doubtful causes,	36 or 18 per cent.

We see from this that hard field service was a large factor in the production of these cases, and next diarrhœa, and thirdly fevers. These are depressant causes, and led largely to the actual production of the malady; but the temperament on the one hand, and the intense excitement on the other, must not be overlooked or undervalued. The American people are, in my experience of them, of a highly nervous temperament, and their nervous system is capable of being strained to a high point of tension. The nature of the struggle, the obstinate character of the fighting, the waste of life, as remarked by military critics, all combined to produce the result. Tobacco does not seem to have played much part in the malady, "some of the worst cases occurring in those who did not use it in any shape." Nor did sexual excess, or the substitutes for it, "produce the disorder, though they predisposed to it, or kept it up." We find it most readily developed in those previously weak and unaccustomed to fatigue, or subject to readily-weakened circulation. (Probably these were persons with congenitally weak or small hearts—J. M. F.) "We find it kept up by irksome equipments and other causes, but not generated by them. (Many cases occurred in cavalry and artillery as well as infantry.) In all these cases it was apt to be noticed that from the outset the double quick was badly borne."

The symptoms of irritable heart are as follows: Palpitation, which differed in individual cases, both in severity and frequency, and considerably, too, was usually accompanied by pain over the heart and in the left shoulder; there was often a good deal of distress; the attacks came on variously, often excited by exertion, but at other times coming on at night in bed. As a rule palpitation was set up by lying on the left side. Pain was almost constantly present; its chief seat was the præcordium, especially near the apex. The pain was not intercostal neuralgia. There was also hyperæsthesia. The pulse was small and rapid, varying from 100 to 140; it was easily compressible. It was powerfully influenced by posture; varying much more than a healthy pulse on assuming the upright posture. When only 80 when lying down, it would mount to 108 when standing; in one case from 84 to 120. The respiration was embarrassed, and shortness of breath complained of; yet it was little hurried; thus the pulse would be 124, respirations 25; pulse 146, respirations 26. Nervous symptoms were complained of, especially headache, giddiness, and disturbed sleep, the last being due to jerkings or unpleasant dreams. Digestive disorders were frequent. It is obvious from these symptoms, thus arranged by Da Costa, that the symptoms of loss of power are blended with nervous disturbance in the irritable heart. Inordinate sweating of the hands was several times complained of; and we know how indicative the wet hand is of nervous exhaustion.

As to the physical signs, "the impulse was almost always extended, yet not correspondingly forcible; rather it is abrupt, or jerky and quick;" there was some hypertrophy commonly; but dilatation was rare. Murmurs were not usually present, and were systolic in time, and generally heard at the apex; thus differing from hæmic murmurs.

As to the diagnosis, it usually was not difficult, dilatation being the condition most approached. Strange to say, phthisis was the condition for which it was most readily mistaken. There is irritative cough, with expectoration, and often hæmoptysis, especially after exertion. "But the aspect of the patient, the pain in the præcordial region, the attacks of palpitation, and absence of the physical signs of tubercle, furnish the distinctive traits." The

condition of irritable heart was sometimes feigned by tying a tight bandage round the lower part of the chest and upper part of the abdomen. When the suspected patient was stripped and told to lie down, and the pulse-rate noted, it was not found to rise high on resuming the erect posture. The impostor usually overlooked the characteristic pain.

As to the prognosis, where the case was due to exhaustion of the sympathetic by hard marching and prolonged excitement, it was generally fairly amenable to treatment, especially when rest was attainable. But in other cases, where the disease assumed the character of a neurosis, the prognosis approached rather that of Graves's disease, and the case was intractable.

As to treatment, Da Costa says: "The treatment is never a short one; and the question arises, would it not be better for the Government at once to discharge these heart cases?" For reasons connected with their service, they were put in the veteran reserve, or put on routine duty; as their discharge would have had a "demoralizing effect." As to the treatment, rest was of the greatest importance; but this the circumstances of the war too often rendered impracticable. Then as to remedies: Digitalis and digitalin "had more influence on the cardiac disorder than any other drug which was resorted to." Then came veratrum viride and belladonna, both of which were useful. Belladonna seemed to exercise great control over the element of irregularity, and often advantageously preceded the administration of digitalis and iron. The latter combination was most useful in cases of debility combined with a very rapid pulse. In other cases aconite was more beneficial. "On purely irritable hearts it had very little effect, nay, repeatedly it was noticed that the impulse became more frequent under its use, and even more abrupt. It was in cases of decided increase in the organ, in cases of hypertrophy, that aconite most showed its influence." He goes on to say, after mentioning its value in apparently arresting organic change, opium as seen in cases where it was given for dysentery and diarrhœa, exercised some quieting effect upon the heart. Hypodermic injections were often used to relieve the cardiac pain, and almost invariably, at least for the time being, accomplished the desired object. Tonics,

as strychnine, quinine, iron, and zinc, were all serviceable in their place, and after the active irritability had been allayed.

"Great care was taken of the men during convalescence. They were mostly placed on guard duty, or other light duty, some still continuing treatment in a modified degree, others not; and were ordered up for examination at stated intervals."

Finally, it may be remarked that the treatment was conducted on two opposite plans—one to increase the heart's contractions by digitalis and its allies, and the other to lower by aconite and drugs of similar action. In those cases where debility was pronounced, cardiac tonics were administered; in the cases where there was excitability with hypertrophy, cardiac depressants were rather indicated.

This condition of irritable heart is not identical with the heart disease found in the British army, and described by Myers and others, where organic changes are set up from the pressure of the uniform and accoutrements.

An increasing experience is teaching me that "irritable heart" is a form of disease with which we are to be more and more familiar; and it is of the greatest importance that it be recognized and distinguished from other cardiac affections. It is a compound of irritability blended with debility, and in the cases I have seen there has been a history of long-continued overwork with anxiety. The persons in whom I have seen it most frequently are medical men themselves. In all there was a history of this kind; a considerable practice involving much anxiety, as well as actual exertion, extending over several years, without any proper holidays. Then came some night work, which of course was of an anxious nature, and a certain loss of sleep which could not be made up from the demands of a large practice. Full, heavy days of work, entailing much anxiety with broken rest, and then the heart becomes irritable. We know that irritability is the precursor, if not the first stage of exhaustion in all nerve tissue; and so it is here. The heart beats rapidly, there are uncomfortable sensations connected with it regionally; its beat is readily accelerated on any effort, and its owner feels less equal to exertion than of yore. It is indeed a neurosal affection, in which there is also impairment of power; the patient is not as good as he once was, or so equal to his work.

In all the cases, too, there was a certain temperament. The neurosal diathesis was pronounced; and the sufferers were men of active habits,—hard-working and conscientious. They all gave me the impression of bearing some of their patients' crosses,—to feel that if Mr. A. died, Mrs. A. and the children would be badly off; they have, indeed, other anxieties on their minds as well as their own. Usually, too, they have been spare men, whatever that may have to do with it. In two cases of American lawyers, who consulted me for overwork and exhaustion, and in whom there was also an irritable heart, both revealed the fact, that they felt intensely about their clients' interests. There is certainly an element of mental worry as a factor in the production of irritable heart, as well as physical overwork. Just as all nervous maladies taking their origin in mental worry combined with too long-sustained physical efforts, are on the increase at the present time, so is the irritable heart; and it is with saddened feelings one reluctantly recognizes the fact that it is just the best of men who are the most liable to suffer therefrom. A cold-hearted selfish man is largely protected by his temperament from being affected with these nervous diseases of modern life. Dr. B. W. Richardson has written eloquently on the subject of "The Diseases of Modern Life," and drawn some pictures which cause one to feel rather hypochondriacal after their perusal; indeed, the pictures are, if anything, too vivid; but probably Dr. Richardson has sketched them from life, as he has long been an authority on the nervous disorders of the heart.

The irritable heart is very intractable, and of all patients medical men themselves are the least satisfactory, from the point of view of treatment. They know too much for their own peace of mind, and though reassured for the time being, the indications of cardiac debility, or excitement, which are part of the malady, soon lead them back to the suspicion of organic disease; and the unfortunate doctor soon conjures up again all his fears and apprehensions. His circumstances, too, are against him; he is in active work with his own cares, as well as other people's; has a family dependent upon him, and cannot give himself the rest requisite for cure. He toils on making the best of matters; sees one consultant after another, too frequently without any satisfactory results; cuts

off what work he can, and continues to live--a crippled being. So far, in my personal experience, no sufferer from irritable heart has died; and this is a very important fact from a diagnostic and prognostic point of view; and makes it imperative upon practitioners to discriminate betwixt irritable heart and the conditions of organic disease, which it simulates. The absence of the evidences of actual organic disease, of dilatation, or fatty degeneration on the one hand, and the facts of the case on the other, the age, the history, and the temperament, will usually suffice to settle the matter. In one case seen recently the cardiac debility was such that the patient had been confined to bed, and then could only take carriage exercise. He had seen Clifford Allbutt, who also took the view that the case was one of debility and muscular weakness, and not of organic disease. He was slowly improving, but the persistence of the cardiac feebleness induced his medical attendant to seek a second opinion. As the opinion given,—quite independent in each case,—was identical, he and his patient felt much relieved and reassured.

As these cases are on the increase the wary practitioner will do well to be on the outlook for them, and not commit himself and make his patient unnecessarily anxious about his case. Our knowledge of the restoration of the heart to its normal size after acute dilatation is comforting in those cases where there might be evidences of slight change of form. Rest is the first thing to be secured, if possible; if complete rest is unattainable, then partial rest must be insisted upon; and a sufficiency of sleep is very essential for improvement. As to remedies, quinine with hydrobromic acid, and in some cases a little digitalis, are indicated. The prognosis is good as to life, but not as to cure.

.Then there are cases met with not quite presenting the features just given, but which are of an allied nature. Thus for instance severe over-prolonged exertion, as mountain-climbing, will bring on a certain amount of cardiac enfeeblement, with paroxysms of palpitation at intervals. Several such cases are known to me from climbing the Righi. An American friend of mine, whom I met in Vienna, told me of an experience of his own. When doing a pedestrian tour in the Tyrol, after a long and arduous ascent of a most perilous character, he felt his head swim on a giddy height;

being a man of iron nerves, he retained complete command over himself during the dangerous descent of the peak. Talking the subject over with his guides he found that they were quite familiar with the phenomenon, and that in some cases the loss of nerve and self-command was such that the victim had often literally to be borne down by the guides. In such cases the severe climb had usually been preceded by something of a depressing character, as diarrhœa, a debauch, or sexual indulgence. Here the condition is one of acute cerebral anæmia, from the imperfect action of the heart. Dr. Clifford Allbutt, in his essay on "The Effect of Overwork and Strain on the Heart and Great Bloodvessels," relates his own experience. After a long day's Alpine climbing a further ascent was attempted. This new effort produced suddenly "a strange and peculiar *besoin de respirer*, accompanied by a very distressing sense of distension, and pulsation in the epigastrium. On placing my hand over my heart, I felt a laboring diffused beat all over the epigastrium." He says: Rest gave relief; but climbing brought it back. After reaching level ground all was well; but about three in the morning he was awakened suddenly with a return of the attack. Here there was acute distension of the heart from effort; and the return of the attack in the small hours of the morning stands in a very suggestive relationship to those cases where persons are found dead in bed after effort the preceding day. Dr. Allbutt may be congratulated that that morning attack was all he ever knew of the overdistension of his heart; for several cases have come under my notice where a sudden effort has been followed by a condition of irritable heart for a considerable time afterwards. In such cases there is an element of muscular enfeeblement—probably chiefly in the right ventricle—present, as well as the neurosal condition.

A condition of irritable heart is not at all uncommonly induced by sexual excess. A friend of mine in Vienna, a gentleman possessed of a physique of more than ordinary vigor, and with good powers, and of strong sexual proclivities, indulged rather freely. The consequence was that the heart's action became feeble, and the pulse small and compressible, but not abnormally fast. This was more marked after indulgence. The condition became chronic; so getting uneasy about it he went off on a walking tour, some-

what doubtful about his capacity to sustain exertion. To his agreeable surprise he soon got a good pulse, became more equal to exertion, until mountain climbing gave him no inconvenience, and every evidence of cardiac debility passed away. On returning to his old practices in Vienna there came back the feeble condition of the heart and pulse, with exacerbations too markedly connected with his indiscretions to leave any doubt about their standing to each other in the relation of cause and effect. Such a condition is found among persons living in the country under the most favorable circumstances, as 'squires and clergymen, and yet who are always complaining of not being well—"not being up to the mark," as they term it. On an accurate analysis it will usually be found that they are rather too attentive to their spousal duties. In others attacks of palpitation follow upon even moderate indulgence.

A condition of irritable heart with impaired power is the common result of a debauch, alcoholic or other. It is of temporary character, and its duration is in proportion to that of the excess which brought it on.

Two toxic agents in common use notably affect the heart, viz., tobacco and tea. Many persons of stalwart physique cannot smoke strong tobacco, from its effects upon the heart. "Smoker's heart" is the term for the irritable condition of the heart brought on by indulgence in tobacco, in use in the Royal Infirmary of Edinburgh. The pulse is not quickened, but is feeble, compressible, and irregular, indicating a condition of cardiac enfeeblement. Distinct intermittency may be produced during the act of smoking. A light tobacco used in moderation will usually be sufficient to enable the individual to avoid "smoker's heart."

Tea produces more active manifestations of cardiac disturbance. There is a condition of irritable heart with paroxysms of active palpitation. Such cases are very common in out-patient hospital practice. The substitution of cocoa for tea usually furnishes relief; but the "tea-drunkard" is very apt to relapse.

An irritable condition of the heart may be set up and maintained by far-away irritation, in the pelvis or elsewhere, in the manner described when discussing palpitation of reflex origin.

Irritable heart with a defective pulse must not be confounded

with the merely "slow pulse." In some persons the pulse is very slow; in the case of a woman sent me by Mr. Pugin Thornton, the pulse-rate was but twenty-four, and he had noticed it as low as eighteen. She had no fainting or vertigo, and her brain was apparently perfectly supplied with blood, though the ventricular halt was long. In a case which was reported to me in Berlin, the young gentleman had been rejected for military service on account of a slow pulse. He was of active habits and a great hunter, and as far as his heart was concerned would probably have been an active warrior, had the opportunity but been offered him. Ordinarily his pulse was forty, and in illness it mounted up to eighty, just the normal rise. Abnormal slowness in sickness is not unknown, and is commonly of no sinister omen. In one family in the north of England this is the case with every member of it. The pulse falls to thirty-five or forty during illness, without any evil consequences; and their condition during health is robust and their life of average duration.

We are rapidly learning the lesson that all heart disorder is not necessarily heart disease. As we have learnt that murmurs do not always indicate valvular disease, so we are learning that there are conditions of cardiac enfeeblement which are not due to dilatation or decay of the walls of the heart, but which are a part of the condition now known as irritable heart—a condition which has to take a conspicuous position in the future.

The vascular system is commonly affected by the emotions. The expressions "stout-hearted," and "faint-hearted," illustrate how the heart may falter in some individuals, and its action be fully maintained in other persons; the "stout-hearted" may be relied upon in an emergency; the "faint-hearted" may faint and certainly lose all self-possession when suddenly called upon. The blush, or Shamroth of the Germans, extends over the whole cutaneous area; it is merely most visible on the neck and face. Some persons can exercise great control over these vascular conditions; the coquette can control the blush, the practiced rifle-shot can keep himself cool, that is, keep down the vascular excitement which would otherwise with bounding pulse shake the steady hand and disturb the aim. A power of voluntary control over the circulation is possessed by certain persons (see p. 36). Dis-

turbance of the heart's rhythm is one of the outcomes of certain cerebral diseases, and is of bad prognostic omen, as in hydrocephalus, for instance. A curious instance of disturbance of the rhythm of heart by cerebral action came under my notice lately in the person of a medical student. He was a tall stalwart fellow, of good family history and excellent physique, but whose heart had aquired the trick of beating unrhythmically, especially when engaged in study. One day when examining him with the stethoscope I asked him a question which necessitated a moment's thought. At once the rhythm was changed, the action was slowed, and there were intermissions. In after visits the experiment was repeated with uniform results. If the question put involved no thought the heart's action remained unaffected; if it entailed thought the rhythm was at once disturbed.

As the "irritable heart" is on the increase, along with the other neurosal conditions of the heart, it becomes important that the diagnosis betwixt it and commencing conditions of cardiac dilatation be established in every case, if possible. Probably it may not be possible to do so in all cases at one interview, and a second or repeated examinations may be requisite in order to make the diagnosis certain. The effects of treatment will often help to clear up the case when the nature of it is obscure. Mere muscular failure in comparatively young and fairly well-nourished persons will be found usually to yield readily to rest, digitalis, and hæmatics; while the irritable heart improves but slowly. The temperament of the patient is also an important factor in the diagnosis.

CHAPTER XV.

THE GOUTY HEART.

FIRST STAGE—LITHIASIS—CARDIO-VASCULAR AND RENAL CHANGES—HIGH ARTERIAL TENSION—DANGERS CONSEQUENT THEREUPON—COMPENSATORY ACTIONS WHEN THE BLOOD IS IMPERFECTLY DEPURATED—SECOND STAGE—HEART FAILURE—CHANGES RESULTANT THEREFROM—TREATMENT—KIDNEY DISEASE FROM HEART DISEASE.

THE association of changes in the heart and arterial system with changes in the structure of the kidney has now long been recognized. James, of Exeter, observed it in 1817. His view was that subsequently announced by Bright, viz., "that the altered quality of the blood might so affect the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system," and hence arose hypertrophy of the left ventricle. Rokitansky recognized the fact that arterial degeneration, true apoplexy, and cardiac hypertrophy were found with chronic renal disease. In 1855, Traube published some writings on the connection (Zusammenhang) betwixt chronic disease of the kidneys and changes in the heart. At first he thought the rise in the general arterial tension due to the contraction of the kidneys and obstructed renal circulation; but he soon gave up this hypothesis as untenable (though it is still given as his mature view by many authors); he then adopted the view of impeded arterial flow, and in 1872 he was fully acquainted with the views of George Johnson as to the thickening of the muscular walls of the arterioles. He held, however, that this muscular thickening was not found in all cases.

In our own country, the work of Richard Bright was carried on by several followers. In 1852, Handfield Jones described a fibroid change in the arteries, which he described as not inflammatory. In 1867, George Johnson described changes in the walls of the arterioles, viz., a thickening of the muscular coat, by which the blood-flow was obstructed. In 1872, Sir William Gull and

Dr. Sutton read a conjoint paper on "Arterio-Capillary Fibrosis," that is, on a change which was a fibrous thickening, rather than an hypertrophy of the muscular wall of the arterioles, and found along with renal fibrosis. They hold this a general fibrosis, in which the kidneys often share, but not necessarily so. They hold that "the contraction and atrophy of the kidneys are but part and parcel of the general morbid change. The kidneys may be but little if at all affected, whilst the morbid change is far advanced in other organs." It is very difficult, in the present state of our knowledge, to take up a definite position on this matter. There is much to be said for both sides. Gull and Sutton seem to have grasped a broader hold than Johnson, while he, so far as he goes, is more precise. Johnson argues truly enough, that it is impossible to account for the high blood-pressure in the arteries on the hypothesis of degenerate arterioles, which would present less, and not a greater obstruction than normal to the blood-flow. On the other hand, there are such general changes found with these vasculo-renal changes, that we cannot help feeling that Gull and Sutton have caught hold of a general change, possibly some premature form of an almost normal senile change, in which the vascular system and the kidneys are commonly, but not necessarily always implicated. It is not essential here to pursue the question as to those cases where the vascular system and the kidneys are not involved. At present the inquiry is confined to those cases where there exists a condition of lithiasis with changes in the kidneys and in the cardio-vascular system, and where the prominent conditions are a tense pulse, indicating high arterial tension and hypertrophy of the heart. So constantly are these found that Dickenson writes: "Since I have had my attention particularly directed to this subject, I have scarcely seen an instance in which, if the renal state was distinctly recognized, whether after death or in life, some degree of cardiac hypertrophy was not also apparent. I have, in fact, got to regard simple cardiac hypertrophy as one of the most important diagnostic signs of renal fibrosis." Rosenstein holds accentuation of the aortic second sound to be highly significant of chronic renal disease. While all observers from Traube to Mahomed have recognized the characteristic hard firm pulse, which gives to the sphygmo-

graph the characteristic square-headed tracing, as being pathognomonic of waste-laden blood with hypertrophy of the left ventricle and chronic renal disease. Indeed the firm, incompressible, steady, slow pulse of this condition should always at once arouse the suspicions of the medical man.

That the muscular walls of the arterioles are thickened in chronic renal changes, as first stated by George Johnson, is corroborated by the researches of Dr. Quain, Professor Rutherford, Dr. Charles Kelly, Professor Garrod, Dr. Broadbent, Dr. Grainger Stewart, Dr. Ringrose Atkins, Dr. Sibson, Dr. Galabin, Dr. Mohamed, and others. Dr. Dickenson says: "The affected vessels, which are apt to be the smaller arterioles, are often thickened in a sufficiently striking manner; this thickening involves both the muscular and fibrous coats. That the muscular coat is increased in bulk, I think, admits of no doubt; nor is it less evident that, with its increase of bulk, it undergoes change of texture." And again: "The regularity with which the arterial repeats itself, in conjunction with the renal state, is such as to show that the parietal thickening generally depends upon constant not accidental conditions. The muscular coat appears usually, though not always, to increase before the fibrous, and in some cases its abnormal bulk is the most noticeable alteration. There are others—and these are cases of long standing—in which degeneration has proceeded to an extent to mark or supersede every other change. The vascular change is associated so constantly with hypertrophy of the left ventricle, that the latter, if independent of valvular disease, or tangible obstruction, may, in a case of renal disease, be generally accepted as evidence of the former." Dr. Galabin found, in sixty-six cases of granular degeneration of the kidneys, no less than fifty-three instances of cardiac hypertrophy, for which there was no other cause that could be discovered.

Some account will now be given of the cardio-vascular changes, which are found along with chronic renal changes. Before doing so, an explanation may be tendered as to title of this chapter. In the first edition, this chapter was headed Combined Heart and Kidney Disease; but this is not lucid enough. In Chapter XIV of the "Practitioner's Handbook of Treatment," the diseases of the heart are described as "primary" and "secondary," the latter

comprising those diseased conditions of the heart, which are found with established kidney disease. But this was also found unsatisfactory; and the Edinburgh title, The Gouty Heart, recommends itself as at once brief and vividly suggestive. It at once embraces the pathology and etiology of the changes, and suggests the therapeutic line of treatment to be adopted.

Such then is the present state of our knowledge; but the advance being made is such that anything written now may not be a fair representation of the subject in a few years. Still it is necessary to describe the condition so far as we now know it. With those cases where the cardio-vascular system is not involved, we are not concerned in the present inquiry; the whole subject will be discussed in a forthcoming work on "Gout in its Protean Aspects," which will be written as soon as the demands of an increasing practice will permit. At present the changes known as "the gouty heart" with its concomitants and sequels are alone being described. For convenience it will be desirable to divide the long pathological process into two divisions; a first stage which will describe the changes which precede and include well-marked cardiac hypertrophy, a tense pulse, hardened arteries, and a large bulk of pale urine of a low sp. gr.; and a second stage, where there is failure of the circulation, a fall in the bulk of urine, and the symptoms of heart failure grafted upon those of chronic renal disease. By such division the subject can be more thoroughly analyzed, and the different factors more clearly differentiated.

As regards the first stage unquestionably a certain progress has been made before the disease comes within the sphere of our cognizance—a certain point reached before we can distinguish any departure from perfect health. We must recognize this fact thoroughly, that the commencement is insidious and hidden from our ken, except where an attack of nephritis has distinctly started the morbid process—which has such a long train of pathological changes and clinical phenomena as its consequences. Granting that it may be questioned whether the kidney changes are always the first step in the general change, it is certain that the changes in the kidneys are usually the first departure from health. A condition of lithiasis is the first link in the pathological chain.

The blood is imperfectly depurated, and the waste products of

albuminoids, undergoing retrograde metamorphosis, are present in it in excess. It has been asserted that the presence of such waste products excites arteriole contraction immediately (Geo. Johnson); while others, as Traube and Ludwig, hold that the arteriole spasm is set up by the effects of the waste-laden blood upon the vaso-motor centre; by which general contraction of the peripheral arterioles is excited.

To the finger and the sphygmograph alike there is furnished evidence of high arterial tension. During periods when the blood is more than usually waste-laden, with or without attacks of gout, the arteries are more than usually tense; and sometimes arteries and veins are both full; the enlarged veins on the hands are turgid, while the radial pulse is hard, full, and incompressible. That there is an hypertrophied condition of the muscular walls of the arterioles, in many cases, is placed beyond doubt by the researches of Geo. Johnson, Grainger Stewart, and Ringrose Atkins. A persistent contraction of these hypertrophied arterioles causes a steady high arterial tension. The outflow from the arteries is obstructed, and so the blood is dammed up in them.

The consequence of this arterial fulness is that the pulse is firm and full in the diastole; and the high blood-pressure in the arteries presents an obstacle to the outflow of the blood from the left ventricle on its systole. The ventricle is imperfectly emptied by its systole; the blood comes in from the pulmonic circulation until the blood-pressure within the ventricle on diastole is abnormally heightened, and muscular hypertrophy is brought about in the manner described in Chapter V. Before the hypertrophy is developed in some cases, there is palpitation; it is commonly found when the hypertrophied heart is undergoing mural decay by fatty degeneration. We have then hypertrophy of the centre of the circulation as well as of the muscular coats of the peripheral arterioles, and a sustained high blood pressure in the arteries.

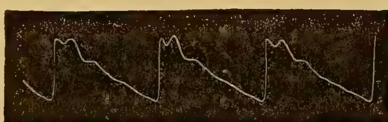
This condition is slowly developed, and up to this point the individual is often the type of health and vigor to look at. The high blood pressure gives vigor and activity to the body generally; the brain, perched at the top of the organism, is well supplied with blood, and its action is energetic and sustained. The complexion is often ruddy, showing a full vascular system; the

walk is firm, showing that the muscles are well fed; the appetite and digestive processes are excellent; indeed, the individual presents the appearance of a well-nourished, active, energetic being, who never has to consult a doctor. In fact it may be affirmed that the active men in middle age, the hard brain-workers of society, are commonly men who are in this first stage of lithiasis, and who in time will furnish the cardio-vascular changes which are the associates of the gouty heart. It does not follow, however, that there are not other persons than the stalwart and ruddy who are in this first stage, and who possess a high arterial tension. Slightly built individuals, of the nervous diathesis, may be rather pallid even, and yet have a sustained high blood pressure in their arteries, and be characterized by energy. The contrast betwixt the ruddy-complexioned persons of the gouty diathesis, and the pallid persons of the nervous diathesis, each being in the early stage of the gouty heart, is considerable; yet comparison soon brings out the points which they present in common.

For years this first stage may go on without evidence of muscular failure in the heart, or of any valvular disease either at the aortic or mitral orifices. Still there are evidences that departures from a perfectly healthy state are going on. The heart is evidently hypertrophied, though it may not always be easy to determine this in consequence of ossified costal cartilages, and some emphysema in the anterior edges of the lung. Its first sound is that of hypertrophy (p. 122), and the second is accentuated. Often this accentuation is very pronounced; and the sound is sometimes of a ringing character, at other times it resembles the sound of the contact of two hard substances; while at other times it is rather of a clanging, or even a booming character. Dr. Sibson taught that the second sound was due not merely to the closure of the semilunar valves, but to tension of the whole root of the aorta, and of the pulmonary artery, *i. e.*, of the sides of the root as well as of the base formed by the valves. This view seems to be well founded, as the second sound at the aortic orifice may at times be heard distinctly through a regurgitant murmur. In some cases there is a certain muffling of the aortic sound, indicating some change in the valve cusps, before an actual murmur is produced. The association of accentuated aortic second

with an hypertrophied left ventricle is very suggestive, especially in a person at or over middle age; and still more if the arteries be full and rigid, or even tortuous. Dickenson thinks the arterial condition so significant that he places it very conspicuously. He says: "Taking first the most marked condition, the pulse with the advanced granular kidney differs from that of health chiefly in this—the tension of the vessel and the distension of the whole arterial system are increased. The systolic upstroke is exaggerated, the diastolic downstroke retarded, and increased pressure is needed to bring out the movements of the vessel. And that that vessel is fuller and tighter than natural is as evident to simple touch as with the instrumental recorder. If in a well-marked case the finger be passed to and fro over the wrist the artery will give the impression of a hard prominent and unvarying cord, like a tendon rather than a vessel."

When atheroma has become pronounced, and especially if it be of a calcareous character, the radial artery may feel rather like a piece of pipe-stem than an artery. In advanced cases it can be rolled about, feeling like a hard tube, often presenting tortuosities. The following sphygmographic tracing is characteristic of renal cardio-vascular change. It is known as "the square-headed tracing" (as is described at p. 82).



When these three conditions are found together, the presence or absence of albumen in the urine is of little diagnostic, though it may be of significant prognostic, importance. Grainger Stewart says, in speaking of cirrhosis of the kidney, "Albumen is rarely present in any considerable quantity, and its presence—fitful in its appearance and varying in its amount—is also difficult of explanation." The bulk of urine is, however, a matter of great diagnostic value. Traube taught, with great distinctness of utterance, that the bulk of urine stands in a strict proportion to the blood-pressure within the arteries. When the arterial tension is high

the pressure in the glomeruli of the kidney is great, and the flow in the tubuli uriniferi swift. When the pressure on these thin-walled glomeruli is lowered, the bulk of urine passed is small. Thus the free flow and large bulk of urine of low specific gravity, which characterizes the early stages of chronic Bright's disease, contrasts in a striking manner with the small bulk of concentrated urine, laden with lithates, and of high specific gravity, which is seen in cardiac dropsy. But as the case of chronic Bright's disease marches on to its later stages of cardiac failure, the changes in the bulk and character of the urine become very instructive; and, as Sir William Jenner has pointed out, the change from the large bulk of pale urine (of the first stage of chronic Bright's disease) to the small dark-colored, lithate-laden urine of the last stage of cardiac failure, is of the highest diagnostic and prognostic value. If along with the other evidences of the later stages of the gouty heart being reached, the bulk of urine is much below what it had previously been, this indication of the fall in the blood-pressure within the arteries is most significant of degeneration in the muscular walls of the heart.

There are certain points connected with the cardio-vascular changes of chronic renal disease, which are of great importance, and are worthy of description. One of these is the slow, steady pulse, so characteristic of this combined condition. Whenever the arteries are full, the pulse is slow. We saw in describing the innervation of the heart that when the roots of the vagus-nerve are flooded with blood, the inhibitory fibres are thrown into action, and the systole of the ventricle is retarded. By this means the blood has time to escape out of the tense arteries by the contracted arterioles, before the next ventricle-ful of blood is thrown into the arteries. If this nerve-arrangement did not exist apoplexy would be infinitely more frequent than it is. If any skepticism exist as to the association of the slow pulse with arteriole contraction, in the mind of any reader, it will at once be dissipated by dropping five or seven drops of nitrite of amyl on a piece of lint, or a handkerchief, and allowing the patient to breathe it. In a few seconds the blush caused by the dilatation of the peripheral arterioles, is accompanied by a complete alteration of the character of the pulse; the slow, hard, incompressible pulse is exchanged

for the rapid, compressible pulse of pyrexia. In a few more seconds this passes away, and the pulse is again slow and hard. Any pyrexia, as an ordinary catarrh, will effect a like but more persisting change in this pulse; and the sphygmographic tracing for the time loses its characteristic square head. The first touch of the patient's pulse should at once arouse the suspicions of the medical man as to the real condition in the early stages of the gouty heart, no matter what the complaint for which he is consulted, and should guide the line of treatment. On more extended scrutiny, it will commonly be found, that the opinion that lithiasis is at the bottom of the trouble complained of is confirmed. For if "the gouty heart" be the most appropriate name for this complex malady from a clinical point of view, "lithiasis" is what it should be regarded from a therapeutical standpoint.

This condition of high arterial tension has sundry direct pathological outcomes of interest. In spite of the inhibitory action of the vagus fibres apoplexy is not uncommon. Many fine stalwart men in the prime of their intellect, if rather past their prime of years, are the subjects of apoplexy in the first stage of the cardio-vascular complications of chronic renal disease.

The blood-pressure is constantly high in the encephalic vessels, especially in those who are intellectually active, and in whom the encephalic blood-current is swift and broad. In such persons the encephalic arteries being highly distended become atheromatous, and consequently liable to rupture. Under two sets of circumstances, such persons have apoplexy. One is the rise in the blood-pressure, produced by contraction of the cutaneous capillaries, brought about by a fall of temperature. Persons with an active circulation (so that they are not depressed by cold, as happens to those who have a feeble circulation) usually feel exhilarated and full of energy on the oncome of cold weather. Such certainly is the case in persons with the gouty heart; but unfortunately this condition, which is due to a high blood-pressure in the encephalic vessels, is accompanied by the risk of rupture of a vessel, undergoing atheromatous changes. The risk of apoplexy overhangs this condition all along, from the early to the final stages; for, even when the heart is far advanced in fatty degeneration, it may still retain power enough to burst an artery, equally rotten, espe-

cially at the seat of some atheromatous patch. An exacerbation of arteriole contraction also may burst an atheromatous artery, as well as bring a decayed heart to a standstill in diastole.

Vomiting is a frequent cause of apoplexy in the early stages, while the heart is strong. The powerful heart forces on the blood in the carotids, while the muscles thrown into action by the act of vomiting compress the jugulars and impede the venous flow; the blood is thus dammed up in the encephalic arteries, and rupture of an artery, commonly the left middle cerebral, follows.

Another cause of exacerbations of the abnormally high blood-pressure, is a more than commonly waste-laden condition of the blood. The blood, highly charged with nitrogenized waste, excites further contraction of the arterioles with their hypertrophied muscular walls, and then follow apoplexy, angina pectoris, or, if the heart is both dilated and hypertrophied, palpitation. One of my patients, a spare man of slight build and pale complexion, whose father died of angina pectoris, consulted me, some years ago, for attacks of angina, to which he had become subject. They were increasing in frequency and severity, until he had become much alarmed. I found that he had put himself upon an almost exclusively meat dietary, under the idea that it would strengthen him, and upon which he had grown rapidly worse. A restriction of the dietary, with a little potash and buchu to cleanse the blood, procured relief in a few days. By regulating his dietary this gentleman has been free from any well-marked attack for years; when he feels any threatenings he drops meat out of his dietary and has a bottle of his mixture, and all goes well with him. Latham with his usual sagacity noticed these associations of angina with conditions of lithiasis. He says, "Simple repletion is soon found capable of bringing on an attack of angina pectoris" in persons such as are now being described, namely, the subjects of lithiasis; especially where there is imperfect digestion. From what we now know of the function of the liver, and the associations of white lithates in the urine, a couple of hours or so after a meal—so well described by J. Henry Bennett, in his work on "Nutrition in Health and Disease"—we can readily comprehend how a quantity of imperfectly oxidized nitrogenized debris may find its way into the blood, and cause a rise of blood-pressure in

the arteries, with all its consequences. Latham says, "I have heard of gout and angina pectoris alternating with each other in the same subject; when there has been more of gout, there has been less of angina; and when more of angina, less of gout." As to the comparative danger of angina pectoris, in the early, and in the later stages of the gouty heart, the reader must refer to Chapter XI.

But all the subjects of the gouty heart, or in other words of the cardio-vascular changes of chronic renal disease, have not got hearts well hypertrophied. As pointed out in Chapter V, in persons imperfectly nourished, the heart is dilated as well as hypertrophied. Such especially is the case with women. When the exacerbation of arteriole contraction comes, whether induced by a low temperature or by an excessively waste-laden condition of the blood, in a person whose heart is both dilated and hypertrophied, the high blood-pressure in the arteries leads to imperfect ventricular contractions, and acute distension of the left ventricle. This condition of the heart produces palpitation often severe. Women, who have got this dilated form of the gouty heart, are subject to be wakened suddenly out of sleep by a paroxysm of palpitation, often accompanied by ventricular halts which alarm them greatly. Such paroxysms are excited by sudden arteriole spasm obstructing the circulation, and producing embarrassment of the heart. In such women palpitation is commonly excited by slight effort. Nor is it difficult to see how and why this is. The heart, not strong at the best, is heavily handicapped by the condition of arteriole contraction against which it has constantly to contend; any little exertion, added to what it has perpetually to contend with, sets up palpitation—the palpitation of muscular adynamy in the gouty heart. Palpitation, then, is one of the direct results of arteriole spasm, to be classed alongside angina pectoris and apoplexy. This form of palpitation, associated with the gouty heart, was well observed by Wardrop, who says: "Besides those persons who have been sufferers from gout in the extremities, and in whom there is no doubt of the connection of the gouty attacks with a disturbed condition of the heart, there are many, and these chiefly women, in whom gouty inflammation appears to affect the heart alone. Indeed, so large has been the

proportions of instances of this description that have come under my own observation, occurring more especially at that period of woman's life when the menstrual flux is about to cease, that arthritic carditis may be considered as a frequent complaint; whilst from the symptoms accompanying it, such affections have been generally arranged amongst *nervous* diseases." It is at and about the menopause that palpitation is so common in women with the gouty heart.

Another outcome of the high-blood pressure of this disease is comparatively rarer than those just described, but is very serious, and that is aneurism. The sustained high arterial tension leads to atheroma, which may be general or in patches. Where atheromatous patches exist, the arterial wall is apt to give way on effort, or on an increase in the blood-pressure, as in arteriole spasm, and then an aneurism forms. Especially is this the case in the robust. For five years I have had under constant observation at the West London Hospital, a color-sergeant, who is the very picture of health, and a fine fellow all round, who cracked his ascending aorta in the efforts necessitated by the autumn manœuvres. When first seen the tumor could be observed pulsating through his waistcoat. Under iodide of potassium combined with a cardio-vascular depressant, either chloral or aconite alternately, it has shrunk till it can scarcely be seen when he is stripped. By this unfortunate accident a very able man is permanently crippled. At other times the tunica intima is pretty evenly affected, and the elasticity of the artery diminished and a brittle condition of the arterial wall produced. Here a blow, especially at the moment of arterial distension, will readily crack the brittle arterial wall and set up aneurism. In the treatment of such aneurism the persistent high blood-pressure within the arteries must ever be borne in mind.

The cardio-vascular associations of chronic Bright's disease march very slowly, if very steadily and irresistibly onwards; and the process is a very chronic one. It is keeping within very reasonable limits to say that the condition may be readily recognizable in persons of forty—whose progenitors have usually lived to be octogenarians, pestered with gout, and ultimately slain by heart disease—who are quite likely themselves to attain great

length of days, and die at last with or of cardiac disease. Especially is this the case in the members of old families, antiquated races. It is quite obvious that the family of a crossing-sweeper is as old as that of the farthest descended Rajpoots, compared with whose genealogies those of our royal family, the Courtenays, and even the typical Welshman, are as matters of yesterday. An old family is a family which knows about its ancestors; and where for many generations there have existed the means to procure more food and drink than the organism actually requires. Under such circumstances gout is steadily developed. It may make regular progress, or advance by fits and starts; kept in abeyance in one generation by steady persistent abstemiousness, but making a bound in the next generation under a less careful regimen. Gradually is gout developed in old families, though it may not manifest itself in the well-known articular form—which though the form best recognized by people generally, and which even the individual himself will admit to be gout, is really one of the less frequent manifestations of lithiasis. The changes in heart, arteries, and kidneys here described are almost a normal senile change in many families, and markedly so in some of the stalwart families of the class known as "country families," and even of those in the next social stratum beneath this. In the two great manufacturing counties of Lancashire and Yorkshire gout has been growing rapidly and extensively, since the application of steam to mills, and the introduction of the steam-engine into manufactures. The great afflux of wealth, which was the resultant effect, led to a generous dietary and the general use of wine, and the consequence is that gout is becoming very common in the third generation of the successful manufacturer. The grandfather lived well, and died old without ever having a touch of gout, unless it was in the muscular pains, which he called "rheumatics." The father lived well, probably knew nothing of doctors; swore at his rheumatism occasionally; had winter bronchitis; and died of aortic disease. The present representative of the family has attacks of articular gout, a great toe that occupies a good deal of his attention, and thickened knuckles—indeed full-blown gout. We are truly passing through a gouty phase of disease; partly the result of affluence, partly

the consequence of the habits of the regency—and what his line of life did for George the Fourth's heart, we shall see presently.

Disease of the aortic valves is common in the class of persons who acquire the gouty heart; and it is my strong conviction that mitral disease of the chronic sclerotic or contracting form, found in elderly persons, especially in the form of regurgitation, is also found with the changes now being described. The strain to which the aortic valves are subjected by the long-continued high arterial tension, is now generally recognized as the cause of aortic valvulitis. The aortic valvulitis of advanced life always is more or less obstructive. There is a development of connective tissue at the base, or insertions of the cusps, and, indeed, around the whole aortic root, from the tension thrown upon it by every aortic systole. This is generally admitted; but the association of mitral disease with a sustained high arterial tension is not so well known. A series of cases have presented themselves to me during the last few years, with mitral disease, where one at first suspected aortic disease. Yet there is no *a priori* difficulty in understanding how mitral disease may be set up as part of the gouty heart. Every time the ventricle strikes, the curtains of the mitral valve are exposed to a pressure equal to the resistance of the whole arterial system—equal to the pressure on the aortic wall. This is greater than that on the aortic cusps, which bear the weight of the arterial recoil only. We know how strain excites aortic valvulitis; we are beginning to recognize its effect upon the mitral vela. The ventricle is hypertrophied and strikes with abnormal force; while the arteries, less elastic than they once were, present greater resistance to the distending wave driven onwards by the powerful ventricle. The consequence is, the strain on the mitral valves is abnormally great, and chronic, or contracting valvulitis follows.

In estimating the prognosis of valvular disease so associated, we must take care to remember that the valvulitis is not the commencement of the disease, with certain pathological changes consequent upon it; we must recognize the valve mischief as part of a large pathological process, of which indeed it is one of the later outcomes. We must note how much of the associated changes in heart-wall and arteries preceded the valvulitis; as well as estimate

how much is the consequence of valve disease. The hypertrophy is not all compensatory muscular growth; a certain amount of it preceded, and, indeed, is causally related to the valvulitis. This subject will receive further attention when the prognosis of heart disease is considered in the final chapter.

The first stage of the gouty heart continues for years before the heart begins to fail. During this long period the individual may enjoy all the sensations of robust health, and no special symptoms of the pathological process, going on within, present themselves. Still the morbid process is progressing with sure and certain step; and in many persons evidences of it are furnished which are of great clinical interest. Leaving the pathological aspect of the condition for awhile, it may tend to make the matter clearer to take it up from the therapeutical aspect of lithiasis. Thus we become engaged for a time with the different outcomes of imperfect renal action. The kidneys become gradually crippled by the slow extension of the morbid growth of connective tissue within them. Dickenson describes the progress of granular kidney in the following words: "The change begins in disproportioned growth, somewhat of a rank luxuriance, in the fibroid tissue with which the arterial channels are uniformly fringed. Hypertrophy of this part leads to atrophy of all the rest. Creeping along the arterial lines with slow and hesitating steps, involving the organ not all at once, but by little and little, the disease makes itself not suddenly, but with so gradual a departure from health, that its date is usually undeterminable, and its existence unsuspected until it has reached an advanced stage." Slowly and step by step the kidneys are being ruined and their functional activity impaired; consequently we get certain modifications in the urine secreted, and certain actions of a compensatory nature, which are apt to be regarded as morbid conditions—as diseases *per se*. These outcomes of the renal impairment are of great interest, clinically as well as pathologically.

The modifications in the urine itself may first engage our attention. The bulk is usually increased markedly, while the specific gravity is low. It is the urine of high arterial tension, and of heart hypertrophy. Where the heart is dilated as well as hypertrophied, and the arterial tension is not very high, then the bulk

of urine is not so great. As to how this change in the bulk of urine is brought about, by the morbid process in the kidney, it would be interesting to discuss; but in the present state of our knowledge the discussion would not be worth the space it would involve. For the present purpose it is enough to say that the bulk is greatly increased, and the specific gravity lowered, until the normal amount of solids are not excreted; and a condition of lithiasis of blood surcharged with water coexists with a profuse flow of urine. There is a certain and positive antagonism betwixt the bulk of urine and the amount of solids excreted. Then in granular kidney the variations in the urine are more marked than in perfect health. In the robust and apparently perfectly healthy subjects of lithiasis the urine is almost constantly copious, pale-colored, and of low specific gravity. In others there may be uric acid crystals found; in no great quantity in most cases. As life advances, and the later stages are approached, pink lithates begin to be common, and may be present in considerable amount. In others the bulk of urine varies from time to time; at one time it is copious and almost limpid; at another time small in quantity and laden with lithates, either white or of brickdust color. Especially is this the case where the action of the liver is imperfect, and the presence of the lithates is associated with imperfect assimilation and dyspepsia. This is more common in the pallid than the florid subjects of lithiasis; in the spare than the corpulent, except the pale fat muscularly-feeble persons of the lymphatic diathesis. Doubtless these changes in the urine are due to vaso-motor disturbances within the kidney (about which we can rather speculate than speak with positive knowledge), combined with variations in the blood-pressure within the arteries. Just as the urine becomes high-colored and laden with pink lithates at the breaking of a common cold in healthy persons, so in some subjects of granular kidney there are intervals when the large bulk of watery urine is exchanged for a small bulk of concentrated urine; from causes too slight and too subtle to be marked as yet, and, therefore, at present inexplicable. The clinical fact, however, remains; and we can understand how, in many cases, the voidance of quantities of lithates gives great relief, being equivalent in its results to an attack of gout; during the flow of copious watery

urine the waste matters had been accumulating within the system. The water being clear is not always a good indication; and at times it is better to see the lithates than not to see them. Something more will be said on this subject when the changes in the kidneys are being considered.

But the kidneys must not be made to bear all the responsibility for these modifications in the urine; it is the liver which furnishes the urea and uric acid, and other products of albuminoid waste. The liver is the furnace in which all waste albuminoids, either from the food directly, from worn-out blood-corpuscles, or tissue-debris, are burnt—oxidized more or less perfectly. Imperfect assimilation, albuminoid food in excess of tissue wants, or functional inactivity of the liver may cause a quantity of nitrogenized waste to accumulate in the blood; if the kidneys are efficient and active then the blood is depurated, and the urine contains the waste, often in urates which are visible as the urine cools. When, however, the kidneys are impaired in functional activity, are congenitally inefficient, or are mutilated by slowly progressing disease, then the blood is imperfectly depurated; though there may be a sediment in the water passed.

The associations of liver and kidney are very close, and in cases of imperfect digestion with a copious sediment in the urine, the doctor of past generations who shook his head significantly, and uttered the oracular word "liver," was not in error; he was only as one who sees through a glass darkly. The constant demand upon the kidney to get rid of nitrogenized waste is the cause, to a great extent at least, of the chronic change in it known as cirrhosis, or granular kidney. In certain cases, notably pallid old women, where the liver is not perfectly healthy, the urine contains some substance which gives it an exceedingly offensive odor, and that, too, not after standing some time, but when voided. In certain cases the starting-point of lithiasis is in the imperfect action of the liver, by which the blood is laden with waste; the kidneys are not equal to complete blood depuration; and then the condition of lithiasis is set up, with all the pathological sequences which follow upon it.

As a consequence of this waste-laden condition of the blood a number of apparently morbid actions are set up in subjects of

lithiasis, which are apt to be regarded as diseases *per se;* a diagnostic error which is serious, as it tends to lead the practitioner away from the real nature of the ailment, and its appropriate treatment. Whenever a patient is seen presenting the following indications of lithiasis, viz., a tense pulse, a loud aortic second sound, with a hypertrophied left ventricle (or may be dilated and hypertrophied both), and passing a considerable quantity of urine, especially getting up at nights to make water, or whose urine is often laden with lithates—then it behooves the practitioner to bear in mind the condition of lithiasis; no matter what the peculiar ailment of which the patient may complain. As the diseases of early life are closely associated with defective nutrition; so the diseases and disorders of advanced life are intimately linked with imperfect blood depuration.

This statement may appear to some readers as at once too didactic and too strongly savoring of antithesis; but it is broadly true. A great number of the recurrent maladies of advanced life are due to a waste-laden blood, and are most satisfactorily treated by measures directed to the general condition. For instance, a medical friend of mine, on entering upon a newly purchased practice, found among his most important patients a lady with an old-standing ulcer of the leg, which for eighteen years had defied the surgical skill of Europe, and had been a cause of no small amount of suffering and annoyance. Talking the matter over one day, and remembering what my surgical teacher in Edinburgh (Joseph Bell) had said about the association of irritable ulcer with gouty conditions, I suggested that the ulcer might have gouty relations. The lady was put upon potash and colchicum, and under simple water dressing this intractable ulcer healed up perfectly in three weeks. Affections of the mucous membranes, as bronchitis or diarrhœa; of serous membranes, as pleurisy or pericarditis; skin affections, as eczema; muscular pains called rheumatism; neuralgia; osseous disease or arthritis—all may be the direct outcome of imperfectly depurated blood—the condition known as lithiasis; or, as Murchison preferred to denominate it, lithæmia. For a clear understanding of the ailments of middle age and advanced life, a good comprehension of the relations of lithiasis is essential. Again and again will the practitioner suc-

ceed in giving relief, or even achieve a cure where others have failed, if he will but bear in mind that what may at first sight be a primary morbid process is really a secondary compensating action. The blood is imperfectly depurated, and the nutrition of some tissues is impaired; at other times different excretory organs are disturbed thereby. Garrod has demonstrated that there are crystals of uric acid deposited in the tissues of the articulations even after a first attack of gout. Bence Jones called chronic Bright's disease a disease of suboxidation (and in one sense he was right), and called various outcomes thereof "compensatory peroxidations." The waste lithates are deposited in the enlarged joints, and inflammations of these joints, with consequent oxidation of much of the waste matter deposited in them, are fairly enough described by him as "the joints for the time being are little supplementary kidneys." It may then be reiterated, that many of the ailments of increasing age are attempts on the part of the system to supplement the action of the kidneys, when insufficient for perfect blood depuration.

The relations of the perspiration to the urine are very interesting and instructive. Carpenter, in an article in Todd and Bowman's "Encyclopædia," entitled "Metastasis of Secretion," points out how, as evolution progresses, the general excretory surface of the lowest and earliest forms of life becomes specialized into areas with particular functions. "The general mucocutaneous surfaces, or some one of its subdivisions or prolongations, is able to take on to some degree the function of another gland whose functions are suspended. This truth was well known to Haller, who asserted that almost all secretions may, under the influence of disease, be formed by each and every organ. This statement, however, needs limitation, and it would probably be safest to restrict it to the *excretions* whose elements pre-exist in the blood, and accumulate there, when the elimination of them by their natural channel is suspended." Just as the kidneys excrete bile when this ceases to find its way into its natural channels, so when the kidney action is insufficient the bronchial lining membrane may secrete acid mucus forming a chronic bronchitis—which may or may not have taken its origin in acute bronchitis; or there may be acid dyspepsia. Skin eruptions are often the

consequence of lithiasis, and the flow of acid is best alleviated by alkaline applications, and the internal administration of lithia and potash. It is, indeed, in its influence upon the therapeutics that the association of lithiasis with certain maladies is so important. Many maladies, which stubbornly resist the ordinary measures, yield readily to a therapeutic attack, which strikes directly at their causal associations. The elimination of urea and other urine solids by the skin is considerable, and where the subjects of liathiasis have a skin which acts freely they are usually little troubled by these intercurrent compensatory actions. Others, however, and they are the majority, possess skins dry, harsh, and imperspirable, so that their defective kidney action is not supplemented by cutaneous activity. It is these relations of allied depurative action betwixt skin and kidneys which makes a chill so dangerous to persons with chronic kidney disease; the crippled kidneys can scarcely depurate the blood under the most favorable circumstances, and are quite unequal to the increased demand upon them, suddenly set up by arrested action of the skin. Constant care must be bestowed by the subjects of lithiasis upon their clothing to preserve health or comparative health, and warm baths and diaphoretics are distinctly indicated after chills.

One topic may here be mentioned in a casual manner, and that is the development of the viscera. It would appear that this is not uniform in all, or constantly proportioned in the individual. Latham speaks of persons with small hearts, who are incapable of much exertion. Murchison speaks of persons with small livers who are liable to bilious attacks, and who are deranged by a little rich food, which ordinary persons could dispose of with impunity. We are all familiar with the small thorax and badly-developed lungs which we so justly dread when phthisis is set up. The imperfect digestive power of some people is also generally recognized; while we all know that some brains break down under tests which present no obstacle to ordinary understandings. Consequently we may fairly suppose that in some persons the kidneys, if not actually small in size, are below the norm as regards their functional activity. It is possible to speculate that the hereditarily gouty have kidneys of this kind; also to raise the hypothesis that persons with good well-acting kidneys do not

readily become the subjects of lithiasis, and are thus enabled to overeat themselves for years with apparent impunity; while other persons are affected by very moderate indulgence. Normally each viscus has a certain amount of spare power in it, so that it is equal to more than the ordinary demand upon it; but this amount of spare power varies greatly in different individuals. It is possible to speculate that in some individuals there are naturally incompetent kidneys, but that a well-acting liver, by producing perfect oxidation of the albuminoids, and a freely-acting skin, getting rid of a large quantity of urea, so diminish the demand upon these kidneys that they are equal to efficient blood depuration. In others, where the liver is not very functionally active, probably active kidneys, passing out large quantities of lithates, so supplement the work of the liver that a condition of lithiasis is warded off. The condition of waste-laden blood leads to a high arterial tension, which is felt in the kidneys as well as elsewhere; and betwixt the high vascularity and functional activity, the blood being laden with nitrogenized debris, the kidneys in time undergo an interstitial growth of connective tissue, which slowly and steadily pervades their structure and destroys their functional activity. The cardio-vascular changes may not always be consequent upon and subsequent to changes in the kidney; it is very probable that in many instances the changes in the circulatory system and in the kidneys are alike the result of a condition standing in a causal relation to each, viz., a condition of lithiasis when the blood is overladen with nitrogenized waste. The practical lesson taught by the study of the pathology of this condition is this,—to reduce to the minimum of tissue-wants the albuminoids taken as food; not a single grain of nitrogen beyond what is absolutely needed should be taken. It is well known that the greatest and most persistent abstemiousness is necessary to avoid gout amidst the members of certain families hereditarily gouty.

Such then is the condition of persons with gouty hearts in their long early stage, and, of course, when the later stages of heart failure are reached, these complications, arising out of an imperfectly depurated blood, are found still more frequently. There are sundry manifestations to which the persons

with lithiasis are subject, in both early and later stages, which are worthy of consideration on this point. One is the localized pain usually felt at or near the lower angle of the scapulæ, a little to the inside, and below the extreme point of the shoulder-blade, on either side. These localized spots of pain, which can be covered by the thumb-end, are always associated with imperfect blood depuration, are said by some to be due to "liver," while others affirm they are connected with "the kidneys." They are found in young subjects who are bilious, or who have lithates in their urine; but they are most commonly met with in older persons who are the subjects of lithiasis. Then there are arteriole modifications worthy of note. The subjects of chronic Bright's disease, often have cold or "dead" hands. There is acute arteriole spasm, and the hands become white, bloodless, and cold, communicating a corpse-like feeling to the touch. When patients complain that their hands often "die," this should at once suggest to the practitioner a suspicion of Bright's disease. At other times there is arteriole dilatation, a condition in my experience only found in women. Women at or after the menopause, who are the subjects of the pathological condition now being described, are often subject to "hot flushes." A deep blush will be seen mounting quickly over the neck and face, lasting a few minutes, and then dying away. During the time the "hot flush," or "hot-fit," as some term it, continues the subject thereof feels very uncomfortable. These arteriole contractions and dilatations are very instructive, taken in connection with the permanent arteriole changes in this condition. What betwixt the waste-laden blood and arteriole spasm impaired nutrition leads to "rheumatic pains," either in the muscles, the sheaths of tendons, or in the joints, and at times to temporary loss of power in the muscles; and in some cases even "wasting" is produced by protracted arteriole spasm.

Sooner or later the nutrition of the gouty heart fails, after which a distinct series of morbid action follows. But during the usually long period which elapses before heart failure is reached, there are many departures from health which are significant, and many morbid actions of great clinical importance, which may

most conveniently be discussed at the present point; after which the further changes in the heart and arteries will be considered.

First are the objective phenomena, which can be recognized by the eye. The subjects of the gouty heart may be broad-built and florid, or they may be spare and pallid; in women there may be obesity along with pallor. The skin may be oleaginous and without wrinkles; vascular, and often with distinct dendritic twigs of dilated vessels. These atheromatous branches of a small artery are very significant, and of high diagnostic value; indicating, as the late Professor Laycock taught, the condition of those parts of the circulation which are shrouded from our sight. Especially are these arterial twigs significant when seen upon the alæ nasi or cheek-bones of a countenance not florid but pallid. In such cases the little twig perforating the skin and running on the surface is usually very atheromatous, and the lumen of the tiny vessels is much increased. In some cases there is an unnatural smoothness of the skin, which glistens so that the unctuous unwrinkled skin is not unlike the smooth surface of the "King Pippin" apple in spring. In the spare individuals, the skin is pallid and often seamed with myriads of wrinkles; "as wrinkled as a John apple," is a popular expression, and the wrinkled, often wizened face is not at all unlike the small yellow, withered and wrinkled, yet sound "John" apple in spring.

The aspect of the temporal artery is also significant. In the florid and well-nourished, it is thick and comparatively soft, as well as tortuous. In others, where the skin is pallid and wrinkled, or at other times (where there is also hepatic cirrhosis) of a discolored parchment appearance, the temporal artery is small and hard, and can be seen meandering much further than is the case in the soft thick form.

The ear, too, presents certain changes worthy of note. In the florid it is usually of a deep red color, with a large glistening tense lobe like a ripe fig. As years go on, the bright red hue may fade, but the characteristic ear remains. In the spare the ear is pale and wrinkled, the lobe looking withered.

The eye may present a more or less perfect arcus senilis, of either the fatty or the calcareous form described at p. 217. There are retinal changes associated with chronic Bright's disease, de-

scribed by Dr. Clifford Allbutt in his well-known work on "The Ophthalmoscope in Medicine." There may be transient blindness as the result of uræmia, passing away completely. Then there may be œdema or effusion into the retina, where the outline of the disk is blurred; or there may be retinal hemorrhage, and, as the clot is absorbed, there may be seen yellow or white patches. These may be more or less extensive, and the effect upon the visual power varies; total blindness is not a common result, but rather impairment of vision. Whatever may be revealed to the ophthalmoscope, there is little presented to the eye, beyond the arcus senilis and the hazy cornea, whose transparency is interfered with from the development of fat-corpuscles throughout its structure.

The teeth are often very significant. Laycock gives as part of the physiognomy of the persons of "the Sanguine Arthritic Diathesis:" "Bloodvessels numerous; heart large and powerful; blood-corpuscles numerous; skin over malar bones highly vascular, giving a floridness to the complexion. Skin fair, firm, oleaginous, perspirable; eyes blue; hair thick, not falling easily; teeth massive, well emamelled, regular, even undecayed in advanced life." The teeth are large and solid in most instances, the lower jaw massive; though at times the teeth are small. But whether small or large, they are not usually affected with caries; they become blunt and worn down in time, and present a very characteristic appearance. Probably the wearing down is largely due to the acid saliva found in the subjects of lithiasis. The expression "gouty teeth," being used by me at the Harveian Society, Dr. Wm. Stewart, at a subsequent meeting in April, 1878, "exhibited casts of the teeth from eleven gouty patients. The patients were not selected for their teeth, but for their gout. They were all solid and thick teeth, worn down at their free edges. In all cases there was a family history of gout. There was a generic resemblance which was unmistakable. Mr. Henry Sewill said that, as a dentist, his attention had been drawn to the teeth of the gouty; he should not say they were pathognomonic, but they were sufficiently marked to form an aid in diagnosis." (Minutes of the Harveian Society.) In some persons with large jaws the teeth are quite massive and very solidly enamelled. The form of the teeth often furnishes a useful clue in diagnosis.

Then the hair is very significant. Fine hair usually falls from the vertex, leaving a bald crown. Thick strong hair does not fall but remains (see p. 218) thick, and is white comparatively early. Iron-gray hair is very suggestive of lithiasis; and in the dead-house of Vienna, diseased kidneys were invariably found where occasional white hairs were scattered here and there amidst perfectly unchanged black hairs. These persons had not died from kidney disease, nor was there any suspicion of kidney implication in life. These isolated perfectly white hairs in a dark head are usually found with other evidences of the early stage of the gouty heart; but a long series of years may elapse before the condition becomes very pronounced.

These external indications are well worth careful study, and are of the greatest value. Very precise observers know well the facial indications of the large white kidney, so allied to the pallor commonly found with interstitial nephritis in Germany; and they can profitably be compared with the florid or withered countenance, which goes with the cirrhotic or gouty kidney common in England.

The subjects of lithiasis, and the gouty heart and kidneys, are very often hale, and, as regards their subjective sensations and capacity to work, healthy individuals, who do not often trouble the doctor. In fact, it may be asserted that this pathological condition is protective against intercurrent diseases. They have a high temperature and do not readily chill, and so are not subject to colds. They are the persons who, Prof. Laycock used to say, may be found in their shirt-sleeves on Arthur's Seat, watching the sun rise on a winter's morning. Bronchitis, as we shall see, they are liable to; and as degenerative changes set in they have winter bronchitis more or less every winter. Many, however, have sundry troubles in consequence of their imperfectly depurated blood, which deserve description. How far these troubles are compensatory actions, supplementing the imperfect kidneys, and how far they are maladies, *per se*, may not be confidently affirmed in every case; but it will be found that whatever the form of disease, it will be materially benefited by a line of treatment which will include the lithiasis.

Gouty skin diseases are common. Eczema is very frequent;

herpes zoster is not at all rare; and the prurigo of old age is well known. Itching of the skin is one of the accompaniments of jaundice, and in lithiasis, the itching is in some cases most intolerable. It is most effectually relieved, as regards local applications, by a strong hot solution of carbonate of soda, with a little bicarbonate of potash, applied twice or thrice a day.

At other times the gastro-intestinal canal is affected. There is a form of acid dyspepsia which yields to potash, and is aggravated by acids, which is common in the subjects of lithiasis. Such dyspepsia is often most serviceable and limits the lithiasis very materially. A dyspeptic old parson once said, "I have been a dyspeptic for fifty years! Thank God for it!" The cause of his pious gratitude was the fact that all his brothers had been cut off by diseases of gouty origin; the dyspeptic alone survived. Then there is "gout at the stomach," of which we know nothing pathologically; as an assemblage of symptoms it seems to be very acute dyspepsia. Sir Thomas Watson suggests that at times it is pork in the stomach. It is possible that it is at times some affection of the great ganglia of the sympathetic induced by the waste-laden blood. In John Hunter's case there were repeated attacks lasting over twenty years. Usually these attacks are found in advanced cases. At other times there is uræmic vomiting, where the vomited matters have a urinous character; and Frerichs claims to have found masses of carbonate of ammonia in the vomited matter. Usually there is also present uræmic diarrhœa, and total or very marked suppression of urine. Carbolic acid, or creasote is very useful in this form of vomiting; but the restoration of the action of the kidneys is the great point to be attended to.

Then diarrhœa is often found with defective kidney action, especially in the amyloid form of disease. In all cases of diarrhœa in the subjects of the gouty heart and kidneys the practitioner must act warily, and not proceed on the assumption that the diarrhœa must be treated as a morbid process, but must investigate the state of the kidneys and the urine. In diarrhœa the bulk of urine is notably diminished, so that it is easy to overlook the fact that the diarrhœa may be a compensating action. If the urine is albuminous it will be well to allow the diarrhœa to run

on until the action of the kidneys is established. To arrest the diarrhœa before this is done is to kill the patient by uræmia. A painfully instructive case of this kind occurred to me on the very threshold of my practice, and formed the subject of a paper on "Uræmic Diarrhœa," read before the Medicine Section of the British Medical Association at Leeds, in 1870. After giving details of the case, the relations of the kidneys and the intestinal canal as to functions were given; and it was pointed out that in cases of kidney embarrassment, diarrhœa is common, the voided contents of the bowels being of a ureous character. In the uræmic condition following scarlatinal nephritis, purgation and the hot-bath are the remedial measures generally adopted. But the spontaneous diarrhœa of uræmic origin in chronic renal disease is not sufficiently recognized.

The recital of the following case may help to put young practitioners on their guard. Though my attention had remained fixed on the uræmic relations of diarrhœa in certain cases, it was not always on the alert as to the effects of appropriate treatment on the prognosis. Some years ago, the writer, when in general practice, was called in to attend an old lady of seventy-two, who had pallor and swelling beneath the lower eyelids, night voidings of urine with great variations in its amount, a dilated heart and a very irritable temper. There was also a history of preceding attacks of nephritis, with albuminuria. She was suffering from a profuse exhausting diarrhœa with almost total suppression of urine; and the small amount that could be collected was slightly albuminous and contained stray granular casts. I decided not to attempt to arrest the diarrhœa until the flow of urine was re-established.

Acetate of potash, spirits of juniper, and buchu were given internally, the skin was acted on by hot bottles in bed, and hot poultices faced with mustard were laid over the loins night and morning, and poultices without the mustard continued during the intervals; beef tea, milk, and gin-and-water formed the patient's sustenance. For two or three days the case went on getting worse, ureous vomiting set in, and the breath had a distinctly urinous odor; the sight became affected; delirium at nights, and a brown-coated tongue indicated the oncome of the typhoid con-

dition; and the patient's children were summoned to take their last farewell of her. Just then an improvement set in, the flow of urine returned, and the diarrhœa ceased; and the relatives, indignant at being summoned unnecessarily, discharged the writer with scant courtesy as unworthy of professional confidence. This was felt to be rather hard under the circumstances; but a consciousness that the prognosis had not been sufficiently guarded would not, however, allow self-pride to assert too positively that the want of confidence was altogether unjustified. The case proceeded uninterruptedly to a good recovery under the gentleman called in; and the lesson was laid by and pondered over. A couple of years afterwards the old lady was seized with diarrhœa, and a medical man, who knew nothing of her previous history, and who held no views as to diarrhœa being a compensating action, was called in; he proceeded at once to arrest the diarrhœa, only too successfully; and the depressed balance never swung back, but remained permanently overthrown, and the old lady rapidly sank. This case is very instructive, especially as to the effect of treatment adapted to the exact pathological condition.

Then hæmorrhoids are common in persons with the gouty heart, and when the arteries are tense, the bleeding often gives great relief. Cases are not very uncommon when the non-appearance of the rhythmic bleeding has been followed by apoplexy; and one case is well known to me, where great dyspnœa, from pulmonic congestion, was immediately and satisfactorily relieved by bleeding from the hæmorrhoids. In the plethoric, it is the wisest plan to let the hæmorrhoids alone.

The serous membranes are liable to become inflamed, and that too recurrently in many cases. A relative of the writer's was for many years subject to attacks of apparently most acute enteritis, with some peritonitis, which frequently seemed as if they must end fatally. When profuse ammoniacal vomiting and purging came on, then relief was at once experienced and improvement set in; comparatively fair health followed for an indefinite period, and then came malaise for some days preceding another eliminative explosion. In the first edition is written: were it not that no attack has yet been fatal, the prognosis would be unhesitatingly given of impending death; but the system seems to have become

habituated to these compensatory disturbances. The old lady survived four years after this was written; but as her years increased, so did the severity of her attacks, and at last a more than usually severe attack carried her off. Her sister also had attacks of uræmic diarrhœa, and ultimately died of uræmia. Pleurisy and pericarditis, the consequence of poisoned blood, are common in lithiasis, while peritonitis is not uncommon.

In a patient with terrible hæmaturia, an attack of pneumonia of the right lung yielded readily to a mixture of potash and juniper in buchu. To what extent pneumonia is connected with lithiasis is not known at present. Bronchitis, on the other hand, is notoriously associated with chronic Bright's disease, especially in the form of winter bronchitis. An acute attack may precede and start up the more chronic form of bronchitis, which is kept up by the constitutional condition. Or the oncome of cold weather, with arrested action of the skin, may and commonly does start up a bronchorrhœa. In summer the skin acts freely, and so supplements the defective action of the kidney that the patient is fairly well; but as the cooler weather comes on the action of the skin is lessened, an autumn diarrhœa appears; then comes the cold of winter, which irritates the bronchial lining membrane, and there is free bronchial secretion. That this rheum is of a compensating character is shown by the fact that the expectorated mucus reddens litmus-paper. There is uric acid in the bronchial secretion in winter, as well as in the perspiration in summer. This winter bronchitis is quite distinct from the bronchial flux of venous congestion found in the most advanced cases in their latest stages. How far true spasmodic asthma is found in gouty persons, as a consequence of the condition of the blood, is not yet known; it is more than suspected, however.

Curious attacks of dyspnœa are, however, not at all rare in the subjects of lithiasis. Basham, who knew them well, called them "inexplicable;" Niemeyer attributed them to œdema of a transient nature; but though this may be so in advanced cases of heart failure, the attacks are too sudden and too transient to be so occasioned in the early stages.

They may be due to spasm of the pulmonary capillaries, or, more probably, to the disturbing effect of the waste-laden blood

upon the respiratory centres. In sleep the respiratory centre acts less energetically than in the waking state, is to some extent depressed in other words, and the waste-laden blood probably further depresses it, till an attack of dyspnœa is the result. In three well-marked cases of such dyspnœa in the subjects of advanced lithiasis, with a history of the regular use of narcotics (opium and chloral) strychnia produced the most beneficial effects. Strychnia is a powerful stimulant to the respiratory centres in the medulla. Belladonna possesses the same effect upon the respiratory centre; and I find these two agents of great utility in the numerous cases of chronic bronchitis with emphysema, and commonly dilatation of the right heart, which present themselves at Victoria Park Hospital in winter. In consequence of the lung impairment and the enfeebled state of the right heart, the temperature of the body falls low in such patients, and it is eminently desirable that they should be put to bed and be kept warm, in order to give the remedies a fair trial. Indeed, such patients are best in bed during cold weather, when their means will admit of it.

Articular disease is not very common in the owners of gouty hearts and kidneys, and is decidedly one of the rarer outcomes of the condition. Still of course it is found, and intermittent attacks of inflammation in joints are termed "the gout," and often clear the system for a considerable period. The appearance of the hands makes the diagnosis to the eye, and needs no description. There is one pathological condition not generally known about the hands of gouty persons, and that is, the condition of a finger being drawn down on the palm by contraction of its flexor tendon, which becomes adherent to its sheath. It has been well described by Sir James Paget. The patient will usually attribute it to the pressure of his walking-stick, or a tool in common use; but a similar condition, usually, however, not so advanced, is to be found in the other hand. This condition is perhaps absolutely pathognomonic of a condition of chronic lithiasis.

It has been said before that the high arterial tension found in the cardio-vascular changes of chronic Bright's disease, in the early stages, endows the individual with much mental energy. There

is, however, one outcome of this state of high arterial tension, and that is insomnia, which is very annoying. The blood-pressure will not fall on getting into bed, and thus permit of that cerebral anæmia which is essential to sleep. For hours sleep will not be wooed, and it keeps aloof till far into the morning, the patient then sleeping for two or three hours, and awaking without feeling refreshed, and in a very irritable state. This sleeplessness is not due to pain, but to inability to get off to sleep; the mind is active, but the thought goes round in profitless and tiresome circles. This condition is accompanied by "fidgets" if the body temperature is high. In such sleeplessness it is well to get up and walk around the room for a brief time, and then to take a draught of cold water before returning to bed. The effect of the cold, both internally and externally, is to first contract the peripheral arterioles, after which they dilate; this general dilatation of the arterioles lowers the arterial tension, and then sleep follows. The temperature is also lowered by these means, and then the "fidgets" are relieved. This insomnia from high blood-pressure is best relieved by chloral; and it is in such cases that the hypnotic quality of chloral is so well illustrated. Opium is the hypnotic for insomnia due to pain; chloral is the agent to employ when the sleeplessness is due to high arterial tension. There are marked alterations in the functional activity of the brain when the high blood-pressure of the early stage gradually passes away, and the opposite condition of low arterial tension, from heart failure, takes its place; these will be considered under the heading of "mental manifestations."

Leaving the clinical aspect of the subject, the pathological changes going on again call the attention. A long and steadily progressive action has been going on in the kidneys during this protracted condition of lithiasis. It is scarcely worth while here to discuss the question whether the cirrhotic condition of the kidneys is in some cases preceded by a stage of enlargement or not; the development of connective tissue progresses throughout the organ—its progress is slow but steady; and probably at times a stride is made, and a comparatively large portion is involved and destroyed by a passing inflammation, sometimes induced by cold. Gradually the kidneys are destroyed and their glandular

elements wasted, until a point is reached, which is incompatible with further existence, and the patient perishes of uræmia, or some of the outcomes of waste-laden blood. During this time the urine varies much in character. In the early stages it is large in bulk and of low specific gravity, and this may persist for years. But when the blood-pressure falls as the hypertrophied left ventricle undergoes fatty degeneration, so the bulk of urine falls too; but as the bulk falls the specific gravity increases. The significance of this has been pointed out at p. 378. Such is the natural history of the disease; but there are peculiarities in the secretion of urine, which call for our attention. That there are nerve disturbances within the kidneys themselves as well as general vasomotor nerve disturbances, we may not doubt; and Clifford Allbutt thinks "worry" one cause of chronic Bright's disease. But as to what these disturbances within the kidney are we are not yet certain. One well-marked symptom there is, and that is the getting up at nights to make water. This has long been recognized as a symptom of chronic kidney disease. Putting aside the effect of taking large quantities of fluids at or before bedtime, there is developed a tendency to secrete urine freely, during the first hours of sleep especially. It is not alike in all, nor is the irritability of the bladder-centres alike in all. One person will get up after two or three hours' sleep and void a large quantity of urine and be disturbed no more; another will be up three or four times during the night; each passing about the same aggregate bulk. This indicates variations of sensibility of the bladder-centres in the cord. The bulk of urine passed in the first stage is large, especially where the pulse is tense. But in those cases where nearly all the indications of lithiasis are present, except the cardio-vascular changes, and the pulse is soft and the heart unchanged in size, then the variations in the bulk of urine, and the necessity to get up through the night are not found. Where the pulse is tense the patient nearly always gets up at night, and to all florid subjects of gouty heart and kidneys the question may safely be put with confidence, "Do you get up at night to pass water?" Where there is much irritability in the bladder-centres the patient may get up at night several times, even when the later stage, with a small bulk of urine, is reached.

The changes in the bulk of urine should always be carefully noted.

At times there is complete suppression of the renal secretion—the *ischuria renalis* of Mason Good. This is in all probability due to vasomotor disturbance within the kidneys themselves—some spasmodic contraction of the renal arterioles. In one case this suppression of urine with uræmic diarrhœa went on for some days; when the urine flowed the diarrhœa ceased; when there was diarrhœa there was suppression of urine. In order to be certain that the urine was not merely retained in the bladder, a catheter was passed during a period of diarrhœa; there was no urine in the bladder, showing it was not secreted. This ischuria renalis will in some instances last for one or two days without any symptoms of uræmia being set up. Cupping over the loins, hydrogogue cathartics, and gentle diuretics are indicated here. If the condition be not relieved, uræmia will be established.

Then the urine varies greatly in character. In health the urine is at times copious and of comparatively low specific gravity; at other times small in bulk, deep in color, and of high specific gravity. In chronic renal changes these alternations become more pronounced. When the bulk is large, lithates are rarely deposited; when small, they are readily seen as the urine cools. They commonly indicate imperfect action of the liver, or an excess of albuminoid food. It is better, however, to see them in the *pot de chambre* than not to see them, when the blood is charged with nitrogenized waste. Disappearance of the wonted lithates in the water is not uncommonly followed by an attack of gout. A shrewd north-country doctor, the late Dr. Pearson, of Penrith, used to give the diagnostic value of these sediments very briefly. "It is all right when you do see them, but the trouble is when you don't see them;" and the lithates had better be visible to the eye than be retained in the blood.

Uric acid is the form in which nitrogenized waste lingers in the blood and tissues. It is an imperfectly oxidized form of waste, and is not nearly so soluble as urea. In the body and blood, it is in combination with some alkaline base, as soda or ammonia. There are points of clinical importance connected with the hue of lithates; and the white urates of defective assimilation differ from

the pink, or at times crimson lithates of imperfect oxidation. In other persons there is a deposit of uric acid crystals in the urine. The full significance of these different conditions is not yet known; but it is important to note the different forms in arranging the treatment. At other times the urine contains oxalates, especially after taking rhubarb, sorrel, asparagus, or tomatoes.

Then tube-casts are found in the urine, and are of great significance. Often it is very difficult to find them, especially in the stage of copious urine. There are various kinds of casts. W. Roberts gives (1) epithelial casts; (2) opaque granular casts; (3) transparent or waxy casts; (4) fatty casts; (5) blood casts, and (6) pus casts. As to their clinical significance, he says: "(1) The deposit may and generally does contain a mixture of two or more varieties of casts and cells. (2) Conclusions as to their pathological meaning must be deduced from the prevailing types rather than from the absence or presence of one or two of a particular character. For example, it must not be assumed that the kidneys are in a state of hopeless fatty degeneration, or even commencing to undergo that change, because one or two cells, or one or two casts display oil molecules. (3) It is necessary, in order to avoid serious error, to examine specimens of urine passed on two or three separate occasions. (4) Bearing in mind these precautions, and having regard to the previous history of the case, the following conclusions are *generally* warranted: (*a*) epithelial casts and blood casts indicate a disease of recent origin; (*b*) transparent large waxy casts, mixed with dark granular casts, indicate a chronic disease; (*c*) epithelium and casts containing much fat indicate fatty degeneration." These tube-casts may be found without appreciable albuminuria. He goes on to say—"The casts *most commonly* seen in chronic Bright's disease are 'small' and 'large' hyaline forms, and 'granular' opaque ones. Any of these may have a few wasted epithelial cells strewed over them. Perfect 'epithelial' casts are rare in chronic cases, and blood casts are still more rare, unless there be concomitant tricuspid regurgitation." Small transparent casts, indicating old-standing disease, and epithelial casts, indicating recent congestion, are found together when the stage of heart failure is reached, and the kidneys are swollen from venous

congestion. Such kidneys present, often very distinctly, the old disease and the new complication side by side.

Albuminuria is not a prominent indication of the gouty kidney, and is seldom present, and then only slight in amount till the last stage of heart failure is reached; and then it is commonly met with as the consequence of venous fulness from enfeebled circulatory power. It will be discussed further on.

The disease progresses insidiously but steadily throughout the kidneys, which become wasted and atrophied; their glandular structure in some advanced cases being almost entirely destroyed, and the tubules obliterated by the compression of the connective tissue upon them. At times the tubules are found crammed with uric acid crystals. These are usually of very dark color, and where there are cysts on the tubuli uriniferi they may be as large as peas. Sometimes they drop into the pelvis of the kidney, forming the nuclei of renal calculi; they may pass from thence into the ureters, either setting up nephritic colic, or finding their way into the bladder they are passed *per urethram,* or remain in the bladder and grow in size.

Dropsy is not usually present until the stage of heart failure is reached. The dropsy of gouty heart and kidneys presents certain characteristics which distinguish it from the dropsy of pure heart failure, as in mitral disease; it will be described in a later section. A certain puffiness of the face, mainly seen only on getting up in the morning; or a certain fulness under the lower eyelid, are the only forms of œdema seen in the earlier stages; but when seen they are terribly significant. They become more pronounced as the case enters on its later stages.

Second Stage—Heart Failure.—How long the first stage of the gouty heart may go on before the second stage is reached may not be affirmed. How far the disease has progressed before it comes to be distinguishable, and how long it has existed before it can be noted, it is not possible to say. We only recognize it after a certain point has been reached. The rate of progress in some cases is much more rapid than in others; especially where syphilis or alcoholism are present. Probably it is not far from the truth to say that in some cases ten years may cover the whole process; while in others probably forty years may elapse betwixt the time

when it would be possible to say that the process was inaugurated, and the final conclusion. Much of course depends on the recognition of the actual condition and the measures adopted. Where the bulk of nitrogenized waste is reduced to the lowest practical minimum, the case has a much better prospect than in others where the dietary goes on unchanged, and the vascular system is kept in a state of high tension, including the renal circulation, and the kidneys are kept in high functional activity by blood surcharged with nitrogenous debris. In time, in many cases, the muscular coats of the arterioles become hypertrophied, and the obstruction offered to the blood-flow is permanent; at times sudden paroxysmal contractions cause acute rises in the blood-pressure within the arteries, and then follow either rupture of an artery, commonly encephalic, or palpitation from acute distension of the left ventricle, or angina pectoris, and probably aneurism at times. The hypertrophy of the left ventricle maintains the high arterial tension; the two hypertrophied muscular ends of the arterial system keep up a high blood-pressure in the arteries; this sustained overdistension leads to atheromatous change, by which the elasticity of the arteries is diminished. We saw in Chapter V how obstruction to the blood-flow produced hypertrophy of the muscular walls of the heart, and as the atheroma advances and the arteries become more rigid and less easily distended, the cardiac hypertrophy is increased. There is that amount which preceded the atheroma, and which is causally related to it; and there is that portion which is subsequent to, and the result of the atheroma. So long as the arterial recoil is good, the supply of blood to the heart itself is kept up, and the tissue-nutrition of the organ is maintained. But as the atheromatous process progresses the arteries become less and less elastic, and their rebound is impaired. Not only so, but the coronary arteries placed at the root of the aortic column are exposed to the full shock of the aortic recoil; they are greatly distended thereby, and become atheromatous, often more markedly so than any other portion of the arterial system. The coronary arteries are thus diminished in calibre as well as rendered tortuous, so that the blood-flow in them is impeded; and so the blood current to the heart is impaired. The changes in the aorta, *i. e.*, loss of elasticity and consequent lessened

recoil, and the changes in the coronary vessels, combine to lower the tissue-nutrition of the hypertrophied heart. At page 198 it is stated that the degenerative changes in the heart-walls will be given at length in this chapter; but they must be more briefly dismissed than was then intended, from considerations of space. The reader must refer back to what is stated at pages 198–9 as to the progress of fatty degeneration throughout the structure of the hypertrophied heart. At first there are but slight indications of the changes going on in the heart-walls. The pulse intermits, or becomes irregular on exertion; the patient is put out of breath on slight exertion; or attacks of palpitation come on from arteriole spasm. Then the ventricle fails to empty itself perfectly on each systole, and so a dilating process is set up. The signs of hypertrophy give way to and blend with those of dilatation; and it is difficult, well-nigh impossible, to diagnose by physical signs alone betwixt this condition of dilatation implanted upon that of pre-existing pure hypertrophy, and a condition of mixed hypertrophy and dilatation which has existed for years, and which is not undergoing any material change. The diagnosis must be made by observing the accompanying arterial changes, the fall in the bulk of urine passed; and, if necessary, by repeated examination of the patient, bearing in mind the effects of well-directed treatment in causing temporary improvement even in hopeless conditions. As the heart fails to fill the arteries, so its own nutrition fails; the elastic rebound is in proportion to the distension; and that is growing less and less as the heart waxes feebler. Imperfect ventricular contraction involves impaired coronary blood-flow and advancing degeneration of the decaying heart-walls. Each portion of the morbid process tells upon the other; everywhere and on all sides the direction is downward. The heart is no longer a source of danger to the arteries; the risk of apoplexy has passed away, giving place to the graver peril of heart failure from fatty degeneration, or mural decay. Side by side, hand in hand, heart and arteries become more and more degenerate, until at last the rotten heart comes to a standstill in diastole,—it can beat no more. The final stage may not be reached until the ravages of disease are most extensive, and the heart is little else than a mass of fat. Where the disease is very extensive, a slight cause, as the fit of

rage in John Hunter's case, is sufficient to bring the heart to a standstill. In other cases a slight muscular effort ends the heart's activity. There may or may not be dropsy. In other cases an attack of angina terminates the case, the decayed heart fails before the obstruction suddenly offered by an increase in arterial tension from arteriole spasm. The blood becomes more highly charged with waste as the failing circulation no longer carries it sufficiently rapidly to the different excretory emunctories; the different tissues are insufficiently fed, while their debris remains unremoved; and so the vicious circle widens in every direction.

As to the actual pathological changes which go on in the gouty heart in its later stages, it may be more instructive to give the post-mortem appearances in one well-marked case than to describe the changes generically.

The case of George IV illustrates the morbid changes in the gouty heart most admirably. He often suffered from the gout, and his luxurious habits of life are well known. When seen by Wardrop, "the King was sitting up in bed, unable to recline in the horizontal posture, breathing with great difficulty, and with an oppressed and intermitting pulse. All the painful gouty feelings which were in the joints had, for some time, subsided, since which the difficulty of breathing had supervened." The King survived two months, and the morbid appearances found at the autopsy are given by Wardrop from the account officially published. They are as follows: "Two pints of water were found in the cavity of the right side, and three pints and three-quarters in the left side of the chest. The left lung was considerably diminished. The lower edge of each lobe of the lungs had a remarkable fringe, which, upon examination, was found to be formed by a deposit of fat. The substance of the lungs had undergone no change of structure; but the mucous membrane lining of the air-tubes was of a dark color, in consequence of its vessels being turgid with blood. The pericardium contained about half an ounce of fluid, but its opposite surfaces in several parts adhered to each other from inflammation at some remote period. Upon the surface of the heart and pericardium there was a large quantity of fat; and the muscular substance of the heart was so tender as to be lacerated by the slightest force. It was much larger than

natural. Its cavities upon the right side presented no unusual appearance; but those on the left side were much dilated, more especially the auricle. The three semilunar valves at the beginning of the aorta were ossified throughout their substance, and the inner coat of that bloodvessel presented an irregular surface, and was in many parts ossified." Here we see there were three distinct morbid changes: (1) Fatty degeneration of the heart-wall, with dilatation of the left ventricle; (2) aortic valvulitis; and (3) atheroma of the aorta; the usual combination in the gouty heart. The heart was much larger than usual, showing that it was hypertrophied when the degenerative changes set in.

The morbid appearances found after death, and the clinical history in life, together with the habits of the individual, combine to furnish an ideally illustrative instance of the gouty heart.

During the period of degenerative change in the hypertrophied heart the atheromatous process goes on in the arteries, which become rigid and inelastic. In the early stages the arteries are tense and incompressible, feeling sometimes almost more like a tendon than a bloodvessel; but as the arterial walls become more and more pronouncedly atheromatous they become hard to the touch, and are usually tortuous. In this advanced stage they exaggerate the pulse caused by the ventricular contraction, and the pulse may feel strong, even when intermissions of irregular character tell that the heart's pulse is waning. From first to last the character of the pulse is most instructive.

There are sundry complications found with the later stages, some of which are the result of the cardio-vascular changes, others of the state of lithiasis, or imperfectly depurated blood. These are so instructive that some tables taken from Rosenstein ("Nierenkrankheiten," 2d edition, 1870) may be given *in extenso*. It may be remarked that he does not say anything as to the structural condition of the walls of the heart in the cases; nor of the valve implicated where there was also valvular disease.

TABLE OF 292 CASES COLLECTED BY FRERICHS.

KIDNEYS.	HEART.	LUNGS.	PLEURA.	PERICARDIUM.
Stage I, 20. Stage II, 139. Stage III, 133.	Hypertrophy, 99. Simple, 42. Combined with valvular disease, 41.	Œdema, 75. Œdema glottidis, 4. Pneumonia, 27. Infarctus Laennecii, 8. Pulmonary gangrene, 3. Tuberculosis, 37. Emphysema vesiculare, 22.	Pleurisy, 35.	Pericarditis, 13.

PERITONEUM.	LIVER.	SPLEEN.	STOMACH AND INTESTINAL CANAL.	BRAIN.
Peritonitis, 33.	Cirrhosis, 26. Fatty degeneration, 19. Carcinoma, 1.	Chronic enlargement, 26. Acute enlargement, 4.	Chronic gastric catarrh, 24. Chronic ulcer, 3. Pyloric carcinoma, 4. Typhoid anæmia, 1. Hyperæmia and gastric catarrh, 34. Tubercular ulceration, 12. Follicular ulceration, 13. Typhoid ulceration, 2.	Apoplexy, 11; 8 with hypertrophy and valvular disease; 2 with atheromatous degeneration. Serous effusion into ventricles or arachnoid, 40. Meningitis, 2. Tubercular meningitis, 1. Tumor of brain, 11.

114 CASES IN THE DANTZIC BOROUGH HOSPITAL BY ROSENSTEIN.

KIDNEYS.	HEART.	LUNGS.	PLEURA.	PERICARDIUM.
Stage I, 12. Stage II, 67. Stage III, 35.	Hypertrophy, 26; 13 with valvular disease. Of these 9 were in the third stage, 2 in the second, and 2 in the first stage of kidney disease.	Œdema, 40. Pneumonia, 25. Abscess, 3. Gangrene, 2. Emphysema, 11.	Pleurisy, 22. Hydro-thorax, 25.	Pericarditis, 17. Hydro-pericardium, 21.

PERITONEUM.	LIVER.	SPLEEN.	STOMACH AND INTESTINAL CANAL.	BRAIN.
Peritonitis, 13.	Fatty degeneration, 19. Nutmeg liver, 11. Cirrhosis, 15. Amyloid degeneration, 3. Simple enlargement, 15.	Chronic enlargement, 32 Recent enlargement, 13. Cirrhosis, 9. Amyloid degeneration, 8.	Chronic gastric catarrh, 12: Œdema of the mucous membrane of stomach and intestines, 17. Follicular catarrh and ulceration of the large intestines, 16. Diphtheria, 3. Catarrh of small intestines, 13. Follicular ulceration of small intestines, 9. Tuberculosis, 1.	Cerebral hæmorrhage, 3; 2 with hypertrophy alone, 1 with valvular disease (combined). Cerebral anæmia, 1. Chronic arachnitis, 6. Serous effusion into arachnoid, 19. Effusion into ventricles, 14. Purulent meningitis, 1.

Kirkes found in twenty-two cases of cerebral apoplexy the kidneys to be small, hard, and granular as a rule, in the fourteen cases where there was also renal disease. The cirrhotic form of renal disease is more common in England than in Germany. In thirteen cases there was hypertrophy of the left ventricle. Eulenburg, in six cases of apoplexy, found cirrhosis of the kidneys and heart hypertrophy in five instances.

A large proportion of these consequent lesions are incompatible with the idea of hypertrophy, and must have been the results of failing circulation. Out of the whole number of 406 cases no less than 125 died in the first stage with hypertrophy; of the other 281 cases no account is given of the heart, though the concomitant or resultant lesions strongly favor the idea that heart degeneration must have been present in many cases.

Dickenson found in 68 cases of granular degeneration of the kidneys simple hypertrophy of the heart in 31, and atheromata in 14, sanguineous apoplexy in 3, and bronchitis in 27. Pericarditis was present in 16, pleurisy in 7, and vomiting, not bloody in 17. An inflammatory condition of the bowels and peritoneum is not very rare, and probably goes with the symptoms given at page 399.

It is evident that the waste-laden blood is the cause of many of the morbid changes given above, especially the effects upon the mucous surfaces and the serous surfaces, including those of the brain.

Uræmia.—Cerebral symptoms, classed under the head of uræmia, are common in the subjects of chronic renal change. Sir Robert Christison thought that the blood was poisoned by excess of urea. Then Frerichs thought that the urea was decomposed into carbonate of ammonia. These two views have both been abandoned. All that is certain is that the blood is surcharged with the histolytic products of albuminoids, derived from the food, or produced as tissue debris. Then Traube evolved the theory of anæmia, with effusion into the brain-substance (Gehirnœdem). In fatal uræmic coma, œdema of the brain is present, with effusion from the arachnoid or the ventricles—a common complication, as the above tables show. It would seem that there is spasmodic contraction of the cerebral arterioles, with effusion into the

perivascular spaces, and from the serous surfaces. In a typical case of uræmia the subject of a clinical lecture by Professor Duchek, in Vienna, the loss of consciousness, the tremors, and incipient convulsions, came and went, were momentary and transient, and were more in harmony with the idea of intermittent spasm than the constant pressure of œdema. When the more advanced condition of persisting coma is reached then œdema is established.

There are often convulsive seizures in uræmia, often preceded by vomiting. In old persons the convulsions often develop slowly, and pass into coma. In younger persons uræmia commonly gives rise to severe headache of a recurrent character.

The breath is often urinous, and crystals of hydrochlorate of ammonia will be developed on a microscopic slide moistened with hydrochloric acid held before the nostrils. The breath-sounds of uræmic coma are hissing in character, contrasting with the snoring stertor of apoplexy. Rosenstein says there is a rise of temperature in uræmia.

A condition of uræmia may be set up by a change of temperature or of wind. In one case of nephritis progressing favorably to recovery the wind suddenly changed to the east, and the air became much colder; this apparently produced a check to the action of the skin, coma came on with convulsions, and, in spite of purging spontaneously set up, the warm-bath and the use of the lancet, which produced temporary, but transient relief, the patient died. As to the etiology of this combined condition Dickenson says: "It is to be noted with uræmia, as with puerperal convulsions, that the seizure may be immediately brought on by mental emotion. In such cases the nervous system is charged almost to explosion by the uræmic irritation; the smallest jar will determine the catastrophe." It would appear that an excess of nitrogen in the blood renders the cerebral cortex more explosive. Brown Sequard found that a highly nitrogenized dietary made his epileptic rabbits more liable to fits; while a non-nitrogenized dietary lessened the tendency; the same has been found in the case of human epileptics. Some are inclined to think that the recurrent attacks of coma are due to rupture of small vessels, one after another. As to apoplexy and serous effusion in the cardio-

vascular changes of the gouty heart, it may be said broadly that apoplexy is associated with the high blood-pressure of the earlier stages; serous effusion with the later stages of heart failure.

The mental manifestations are of great interest. In the first stage, that of high arterial tension, the brain works well under the high blood-pressure, and the mental processes are active and vigorous. But with this there is a certain amount of irritability, and explosiveness of temper. The irascibility of persons suffering under an attack of acute gout is well known. According to the personal experience of Sydenham there is also mental depression present. Even when there is no articular gout present the temper is apt to be impaired when the blood is unusually charged with gout poison. And this, perhaps, is the most constantly present of all the indications and symptoms of a gouty state of the blood. The annoyance caused by small trifles is out of all proportion to the exciting cause. The patient is conscious of this state of irascibility, which adds to the irritability.

A near relative of the writer's used to say, when in such a condition, that she "could fight with a feather." In such states these sufferers are readily "put out" by trifles; they are irritable, fault-finding, and quarrelsome, approaching the state which Americans speak of as "pure cussedness." This is due to the irritation set up by the poisoned blood, and Bence Jones, in his "Physiological Essays," thinks that it is the presence of oxalates in the blood which produces this mental explosiveness. This condition is chiefly associated with the secretion of a large quantity of pale urine of low specific gravity, and is probably due to a very high blood-pressure, with blood unusually charged with waste, the condition being relieved when the arteriole spasm yields, and the urine falls in bulk, but is densely saturated with lithates. Pain, with a sense of weight at the vertex, is the indication of cerebral anæmia (whether due to peripheral arteriole spasm or to a general state of anæmia, it matters not), and it is accompanied by a general sense of depression and unhappiness. A sense of misery is, according to Maudsley, the evidence of the brain being badly supplied with blood. When the heart begins to fail and the brain is badly fed with blood, the mental manifestations change. Mental vigor gives way to irritability, petu-

lance, caprice, vacillation, unreasonableness, obstinacy, and often ill-nature. So afflicted, the unfortunate individual is the plague of their lives to all with whom he comes in contact. It is impossible to keep quiet; the mental irritability must have something on which to expend itself. Such persons often find the cares of a business or a profession intolerable, and retire therefrom; unfortunately, the repose they seek is not often so satisfying as they hoped for, and they find their way on to boards and committees, where they form a simple unmitigated nuisance, their mental irritability taking the form of mischievous perversity, or of ill-controlled interference with everything and everybody. In this condition they remind the writer of nothing so much as a cancerous gland—no longer fulfilling any useful purpose, but constituting a source of irritation to everything around them. Such persons are often the terror of their households, and of every one dependent upon them. George Eliot has described the mental condition of Peter Featherstone, in "Middlemarch," with the greatest skill and the utmost fidelity; his naturally perverse and wilful character is modified by his physical state, and the whole is sketched so accurately that every medical student ought to study the case. Such a mental condition often strains to the utmost point of tension the relations betwixt patient and medical attendant, while the latter has often to be a peacemaker betwixt the irascible, unreasonable, and withal suspicious patient and his nurses and others around him. Sometimes there are positive hallucinations, of a vivid and distressing character. The patient wakens up suddenly, and the subject-matter of the disagreeable dream is projected into the waking consciousness, so that it becomes difficult to dispel the impression that it is an hallucination, and not something actually seen. Such hallucinations of the most vivid character occurred to one patient of mine, an old gentleman with a gouty heart in the later stages; they preceded acute fatal brain symptoms. All through they were characterized by suspicion, and at the last the patient suspected poison and refused all nourishment.

As the heart fails, whether there be valvular disease present or not, we get the symptoms of heart failure given in Chapter IV, grafted upon those of the early stage; the high arterial tension

gives way to venous fulness; the tense pulse is exchanged for one irregular in force and often impaired in rhythm; the large bulk of urine dwindles down to a scanty saturated urine, of high specific gravity and laden with lithates—the characteristic urine of heart failure. The arterial system is no longer full of blood; the tissues are imperfectly nourished. Not only are the arterioles empty, but the venules are full. The nutrition becomes impaired, and the different tissues become decayed.

We have seen in the above tables the consequence of a failing circulation with a waste-laden blood upon the different viscera and serous and mucous surfaces. Especially are the tissues of the lung and the lining membrane of the bronchi affected. Emphysema is found with chronic bronchitis, and commonly with albuminuria, while the right ventricle gives evidence of failing power. The rotten lung-tissues give way and tear under the repeated cough; the patient's features are cyanosed; the breathing is labored, the accessory muscles of inspiration are extensively thrown into action, while the pulse is feeble and irregular. Basham has described the general condition of tissue rottenness in these cases, and delineated the appearances in the following plate, taken from his work on Dropsy.

DESCRIPTION OF PLATE II.

Fig. 1 is a portion of the vesicular structure of an emphysematous lung, showing the numerous fat-grains deposited in it.

Fig. 2 represents sections of the bronchial mucous membrane, showing the successive layers of cells degenerating as they approach the free surface to be thrown off as mucus, and pus-corpuscles mixed with large granule-cells, and aggregations of disintegrated nuclei represented in Figs. 3 and 4.

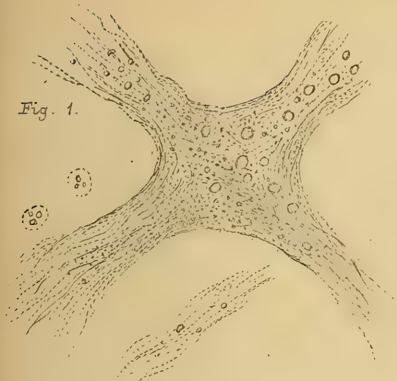
Fig. 5 illustrates the fatty condition of the coats of a small artery leading to an emphysematous patch.

Fig. 6 represents a state of fatty degeneration of the muscular fibre of both auricle and ventricle of the right side, and is typical of what may be seen in most cases of dropsy with dilatation of the right cavities, complicated with emphysema and chronic bronchitis.

From this it will be seen that the degenerative changes are extensive throughout the issues.

This plate (No. XVI in his book) "represents the fatty decay of the tissues observed in cases of emphysema and chronic bronchitis terminating in dropsy."

Fig. 1.

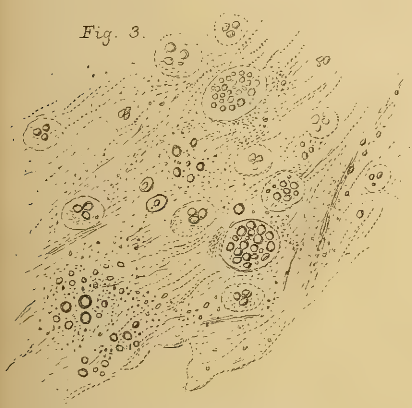


Pulmonary tissue from emphysematous lung.

Fig. 2.

Bronchial mucous membrane.

Fig. 3.



Sputa; Chronic Bronchitis with Dropsy.

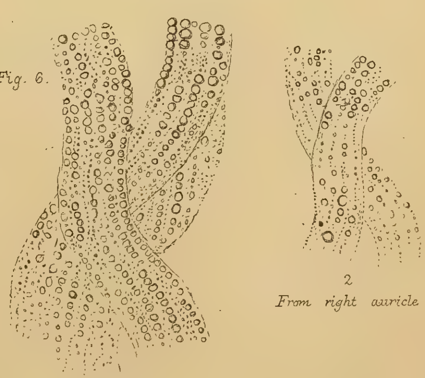
Fig. 4.

Sputa; Emphysema.

Fig. 5.

Fatty condition of small artery adjoining an emphysematous patch.

Fig. 6.



From right ventricle

From right auricle

Heart fibre.

W.R.B. ad nat. del. H. K. Lewis Publisher, 136 Gower Street

There is general venous congestion which leads to still more imperfect blood depuration. The difficulty in breathing becomes aggravated at times, until an asthmatic state is simulated. The imperfectly depurated blood leads to further vasomotor disturbance and arteriole spasm, so that the failing and embarrassed heart is still further handicapped. Attacks of heart failure manifest themselves. Further complications are set up; dyspepsia from urine salts is now supplemented by gastric catarrh; there is serous effusion from the mucous lining of the intestines with diarrhœa; vomiting is frequent; venous congestion leads to enlargement of the liver, spleen, and kidneys, which are all in a cirrhotic state. Bilious derangement comes on frequently. There is drowsiness with unpleasant dreams or vivid hallucinations; and the patient's condition becomes pitiable in the extreme.

The urine presents interesting modifications. The changes in bulk and specific gravity have been spoken of before; now something may be said about albumen in the urine. In the early stages of the gouty kidney, albumen is only present in the urine in small quantities and at long intervals. As it is not associated with healthy kidney structure, nor yet with that portion irretrievably destroyed, we are driven to the conclusion that albumen in the urine here indicates a new piece of kidney structure being destroyed. This is rendered the more probable that it is present fitfully and in small quantities. But when the stage of distinct heart failure is reached, then albuminuria becomes established and is persistently present, as in the advanced stage of heart failure in mitral disease. It is that form of albuminuria which is caused by fulness of the renal veins. Ligature of the aorta below the renal branches does not produce albuminuria; but ligature of the renal veins does set it up. When then the heart fails and the tricuspid leaks, albumen commences to show itself in the urine. It may not be constant at first, or it may disappear for a time under appropriate treatment; but its appearance is ominous. The absence of albuminuria in the early stages gives no comfort, and does not overthrow the other evidence or disprove the diagnosis; but its appearance in the later stages is most significant. Late albuminuria with the appearance of exudation-casts tells that the end is not far off.

Dropsy is not found in the early stages of the gouty heart and kidneys; but it frequently manifests itself while the heart still possesses much vigor and the pulse is tense. It differs from pure cardiac dropsy in that it does not appear in the lower extremities only; the arms are usually involved at an early period. The puffiness under the lower eyelids with a general blurred outline of the features, which is seen long before any other œdema manifests itself, is very significant. But usually years elapse before other œdema is set up. And when it does show itself, frequently the bulk of urine is considerable; while the heart's action is good and the pulse firm. How anasarca is set up under these circumstances, it is rather difficult to say; but there is no question as to the fact. It has been suggested that the amount of urea in solution causes ready effusion from free surfaces and into the areolar tissue. Syer Bristowe says of the retained or non-eliminated water in chronic kidney disease: "A great deal no doubt passes into the connective tissue and accumulates there, the capillary system throughout the body acting as a kind of general secretory organ, and hence results general dropsy." And with the statement of the clinical fact, we must rest satisfied at present. Such cases of cardio-renal dropsy are those in which Withering found digitalis to be of no service (p. 281), and the experience of others is to the same effect. Purgatives, diaphoretics, the warm bath in any form, are the measures best adapted for the removal of this form of dropsy. One point must be insisted upon; incisions, however made, are of far greater service in cardio-renal than in uncomplicated cardiac dropsy. The dropsy of the gouty heart and kidneys will often be cured by the measures mentioned, and remain away for many months, to return when intercurrent disease has depressed the patient; again it will be removed and stay away, and the patient may die with the limbs entirely free from œdema. Southey's drainage-tubes are excellent. They, or incisions which scarcely relieve pure dropsy from primary heart disease, are of the greatest value in cardio-renal dropsy.

As to the sex in which these vasculo-renal changes manifest themselves, undoubtedly they are much more common in men; but well-marked instances are seen at times in women. But the diagnosis in no way rests on the sex of the patient.

As to the age at which these changes are seen, there can be no question. Advancing age increases the liability to chronic degenerative changes.

As said at the commencement of this chapter the change is one allied to senile decay, and brought about more or less prematurely in different individuals and families. Syphilis and alcoholic excess hasten it undoubtedly. So, probably, does the inherited constitution in many cases. An attack of nephritis may start this change in some cases; while in others the change seems well established before any evidence is furnished that the kidneys are specially involved. And this holds good of the cases, seen occasionally, where the cardio-vascular changes are found well marked and unmistakable in young subjects, that is, persons under thirty-five. It seems strange that when these changes are found in young persons, it is in women that they are chiefly found. In such a case in a patient at Victoria Park Hospital, the tense arteries, the hypertrophied heart, and the loudly accentuated aortic second sound, were found in a slight little woman in whom there was no history, and indeed little suspicion of either syphilis or alcoholism. Doubtless in the majority of cases the changes have existed for some time before any evidence of them is furnished, *i. e.*, before they come within the sphere of the cognizance of the physician. Indeed up to a certain point the diagnosis is rather a matter of surmise than of certainty. Much depends upon the skill and knowledge of the medical man. There is something learned by experience which is quite incommunicable from individual to individual; a juggler who can keep six balls in the air at once can easily show one how he does it, but that does not enable a neophyte to do it. He must learn to do it, or to come near doing it, by sustained efforts of his own. The eye, as the old Italian painters said, can only see what it has learned to see.

As to the diagnosis of this condition it is a matter of some difficulty until the whole subject has been carefully thought out; after that it is quite easy. After the matter has been worked at, the *tout ensemble* becomes so clear that the eye almost is sufficient to make the diagnosis; and examination merely corroborates the opinion formed from what is seen. At times, however, the eye is "out," and all the external appearances which have been de-

scribed are found with soft arteries and a normal heart. In such cases there is often dyspepsia and a freely acting skin, itself liable to disease, especially eczema. It is well to note such cases and to learn that a condition of marked lithiasis may exist for years and yet the cardio-vascular system be in no way involved. In others the cardio-vascular system seems to bear the brunt of the lithiasis. It is of course with this last division we are concerned here; nevertheless it is well to make a note of the other division, and to contrast the two. Tenseness of the pulse in the first stages, and atheroma of the arteries in the later stages, are the two leading diagnostic indications. Rosenstein places the heart changes first; for a certain amount of hypertrophy of the left ventricle is requisite to maintain the arterial tension. There is a tense pulse, the temporal arteries are conspicuous, tortuous, or thickened. The eye, ear, and complexion in the florid and the pallid have been described. In some cases there are otolites, which when seen are very significant. There are variations in the bulk and character of the urine, and the getting up at night to make water, which is of the greatest diagnostic service. Albumen in the urine is instructive when found; while tube-casts furnish most valuable information. The change in the bulk and character of the urine, the large flow of low specific gravity of the early stage passing insensibly into the scanty concentrated urine of heart failure has a prognostic as well as a diagnostic value. Aortic valvulitis may or may not show itself; it is not an essential factor. There may be palpitation, especially in women, where the hypertrophy is combined with dilatation, and where the hypertrophy is failing from degenerative decay. It may be found in cases where the cardio-vascular change is but little marked, when the blood is more than usually laden with waste. There is irritability of temper, one of the commonest symptoms which varies with the condition of the blood. Sleeplessness is common. There is disturbance of vision. There are changes in the respiratory organs, chronic bronchitis and emphysema being commonly present in the later stages. Attacks of dyspnœa are not unfrequently found. There are arteriole disturbances, as "dead" hands and feet; and flushes in women at the menopause. There is an accentuated aortic second sound and a free flow of urine; the evidences of high arterial tension. There

is atheroma of the arteries steadily developed. Then there are compensating actions, as diarrhœa or uræmic vomiting. There are inflammations of the serous membranes, and diseases of the skin. There is often articular gout quite unmistakable. There are uræmic attacks, headache or coma, occasionally found. The hair and teeth often give valuable aid in diagnosis. The diagnosis indeed is a complex affair; and the sphygmograph, or better still the trained finger and the stethoscope are more important than the reaction of the urine in a test-tube; for the diagnosis of gouty heart and kidneys rests but little on the presence or absence of albumen in the urine. Recurrent attacks of rheumatism are as suggestive as winter bronchitis.

"There is no drain of albumen in the urine in these cases; its appearance is but fitful and transitory, till the stage of heart failure and venous engorgement is reached. This absence of albuminuria is important, as too many are inclined to regard the kidneys as sound if they can detect no albumen after repeated examination. Such inference is unwarrantable. I may briefly relate a case in point. My colleague, Mr. Teevan, was requested to perform lithotomy on a gentleman from South Africa who had a tumor in his abdomen. He desired me to examine the man prior to operation. I passed over the tumor as unimportant, but strongly advised no operation, stating it to be my opinion that the patient had chronic heart and kidney changes. This opinion was represented to the patient and his friends, but the patient begged to be operated upon. He was cut and died. His kidneys were shrunken and contracted, till scarcely a sound piece could be found; one weighed one ounce and three-quarters, the other, three and a quarter. The heart weighed fifteen ounces, and was fatty. Such is the report of the post-mortem, which I did not see. The letter which the patient brought with him from his medical attendant at the Cape, stated that repeated examination of the urine, chemically, and by the microscope, gave no trace of albumen or casts; and on this negative evidence, the kidneys were assumed to be sound. As the event proved, this opinion was utterly erroneous. The man's general appearance and the presence of the symptoms detailed in an early portion of this paper, furnished positive and trustworthy evidence as to the

actual state of the viscera." (From a paper read by the writer before the Midland Medical Society, November, 1876, which afterwards appeared in the "Birmingham Medical Review," entitled "The Relations of Gout to Disease of the Heart.")

The prognosis is a much more difficult matter. Except in those cases which occur in young subjects, the prognosis as to life in the early stages is good. Indeed it would scarcely be going too far to say that the condition of the gouty heart and kidneys is rather protective against ordinary maladies. The gouty are comparatively free from ailments not directly connected with the condition of lithiasis. Any intercurrent disease, as bronchitis for instance, will tend to become more or less chronic, from its furnishing a local habitation for the lithiasis to settle in and manifest itself. An injury may determine an outbreak of gout; or lithiasis may pitch upon the seat of an old injury and cause persisting "rheumatism" there. Then there are intercurrent disturbances of a depurative order which cleanse the blood, as articular gout, or an outbreak of eczema. They create more disturbance and alarm in persons who are hale and rarely ill than in those who become habituated to these recurrent depurative processes. Apoplexy, angina pectoris, and aneurism are diseases which rest for their production upon the high arterial tension, the high internal blood-pressure of this condition, and are sources of grave danger to life. In consequence of the integrity of the kidneys being cut down by progressive disease, all persistent pyrexia is terribly dangerous in cases of renal cirrhosis; and in the subjects of chronic Bright's disease a temperature of 101° would alarm one greatly. Murchison has found the subjects of cardio-renal changes to die readily in fever. The impaired kidneys are unequal to the demand upon them where pyrexia is melting down the nitrogenized tissues rapidly, and the blood is laden to excess with albuminoid waste matters. Yet in some cases the brown tongue and sordes on the teeth, and a soporose condition, indeed a commencing state of uræmia, are comparatively free from danger; the casual observer would diagnose impending dissolution; the old family attendant is much less alarmed: he has seen it before and hopes that once more it will pass away. He "knows the patient's constitution" and gives a

hopeful prognosis, when to a stranger there is not left a ray of hope. He may not know much about the pathological changes of this complex state; he is in a state of primitive ignorance about compensatory actions, peroxidizing processes; but he has learned from experience to know that these attacks are not only recovered from, but that the patient feels unusually well for some time after them; like thunderstorms which clear the air they possess a depurating action upon the waste-laden blood.

The compensatory action or inflammatory depurative process will bring the patient to death's door, and leave him there; and just as all hope must apparently be abandoned, improvement sets in, and goes on to convalescence. The condition resembles a depressed balance, the lowest point is reached immediately before the upward rise commences. Such conditions are very puzzling to strangers, and even those familiar with the patient recognize the imminent danger inseparable from the uræmic or typhoid state, and are unable to tell when and in which attack the balance will be permanently overthrown. The same holds good of attacks of bronchitis, the patient sometimes recovering from an attack which apparently must be fatal. Here the structural integrity of the muscular fibres of the right heart is the important factor.

When an advanced condition is reached, the subject of chronic heart and renal changes lives, as it were, over a volcano, whose sudden explosions are a source of imminent danger; a fall of temperature, a sudden accumulation of waste in the blood, may send up the blood-pressure in the arteries with all the dangers consequent thereupon. A sudden fit of intense cold will throw a number of these patients on their doctor's hands, and the death rate among them will be very high.

The progress and termination of the gouty heart is as unpleasant a subject for contemplation as medicine can furnish. The only thing to be rationally hoped for is sudden death. The progress is steadily downward and the vicious circle ever widening; and new miseries are added to a load already felt intolerable. An attack of bronchitis survived leaves more emphysema, more extensively lacerated lungs, with greater dyspnœa. Persistent diffi-

culty of breathing is aggravated by more frequently recurring asthmatic paroxysms. Sleep is disturbed by terror-inspiring dreams, which awaken the patient and cause him to dread sleep. It is difficult to say whether the waking or the sleeping condition is the more trying to the patient. The future contains no hope; the retrospective of the past is embittered by the memory of miseries survived. The long vista of the patient's memory is crowded with unpleasant experiences, since first an imperfectly depurated blood led to mechanical results; these in their turn leading to morbid consequences in various viscera. One new trouble is added to another, the patient saying, "This new ailment frightens me so; I had got accustomed to the old one!" The condition reminds one of Dante's Inferno, where as soon as habitude had rendered one condition of misery somewhat less intolerable than at first, another is supposited with new tortures. The brain fed with blood robbed of its albumen and poisoned with urine salts, and but sparingly supplied by a failing heart and rigid arteries, and oppressed by venous congestion, falters; the intellect is impaired, the will is sapped, the mind becomes oblivious to the outer world, and the attention is centred in the physical condition alone; the world shrinks to the dimensions of the sick-room. The world no longer possesses any attractions for the patient; and when the last final scene is reached, the impression remaining in the minds of the survivors, is rather that of trouble removed, suffering ended, than an acute sense of loss sustained.

Treatment.—The treatment of the gouty heart follows the pathology of it. There is the treatment of the early stage previous to heart failure; and the management of the later stage, when the failing circulation has to be included therein. The first point is to remember that the presence of nitrogenized waste, in excess in the blood, is the initial departure from health. Consequently the dietary is of primary importance, and not one grain of nitrogen beyond the absolute wants of the system should be taken in the food. Gouty persons do not require to have their blood surcharged with nitrogenized waste, in order to be mentally energetic; as is the case with those languid personages of the lymphatic diathesis. Little brown meat should be taken; cheese and

eggs should be sparingly indulged in. The dietary should consist of foods beginning with F,—fat, fish, fruit, and farinaceous foods. A lobster salad is the typical dish for the gouty man with a perfect digestion—without a powerful digestion it is poison, producing acute indigestion. Boiled fish is better than fried fish where the digestive powers are feeble. Oyster soup followed by stewed fruit and cream is a sufficient lunch for a gouty man; for the less opulent, some mashed potatoes well buttered, with pepper and salt, set before the fire to brown, form an excellent lunch, with a glass of wine or a draught of milk. Dinner should not, as a rule, include a solid joint, and fruit should always be eaten after it. A digestive biscuit is enough for supper. Breakfast should consist of a rasher of fat bacon, some fresh fish, or cold fish done *au gratin*, or a plate of porridge; where the digestion is feeble, and the life led entails little toil, mental or bodily, a diet of lentils, either whole or ground, and fruit is sufficient. Even when there is a drain of albumen, it is not necessary to put the patient upon a highly albuminoid dietary; very often the drain indicates excess of albumen and passes away, more or less entirely, when a non-albuminoid dietary is insisted upon. An impression exists in my mind, not founded solely on a personal experience, that vegetable albuminoids suit the gouty better than animal albuminoids; but this must go for what it is worth. Then as to the use of alcohol. Many practitioners forbid malt liquors absolutely and in all cases. But my experience goes to teach me that in some cases malt liquors are not contraindicated if the dietary be properly arranged. As to wines, the better their quality the more dangerous they are; for most gouty persons a poor thin claret is the only beverage—whatever wines they may set before their guests. A pure spirit with seltzer or potash water should be their drink to their cigar, or when not eating. Alkaline waters, Vichy, Vals, Carlsbad, Buxton, Bladon (A a), and Congress Springs (Ca.), are always good; and a draught of such water with or without some purgative, as Pullna, Frederickshall, Hunyadi Jànos, or Estill (Ky.) water, first thing in the morning on getting out of bed, is good. Even a draught of plain water taken then washes the tissues of the body and removes debris, before the blood once more contains the products of assimilation; but when

the water contains alkalies and is also purgative, it is better, especially for free livers.

Or others may prefer some good granulated effervescent drink, as granular citrate of potash, or citric acid with a mixture of potash and soda with a little lithia. There should be a certain quantity taken daily to meet the continuous formation of uric acid in the body. Visits to spas in various parts often give great relief to gouty states.

Then as to the use of medicines. Digitalis is rarely indicated in the early stages, except when there is cardiac dilatation, though useful enough in the second stages. Potash sometimes, however, is very depressant, and then the combination of a little digitalis, or nux vomica, with it antagonizes the depressant effect. When there is palpitation, potash, freely diluted, with buchu, will often relieve more effectually than digitalis. As to the use of colchicum, it certainly often gives great relief; but there are many old gouty persons who fervently wish they had never touched it. Personally, it appears more desirable to regulate the dietary than to palliate matters by the exhibition of colchicum. A mixture of potash bicarbonate, or Rochelle salts, with a little nux vomica in buchu, is my favorite prescription for gouty patients. Iodide of potassium at times proves good when the bicarbonate alone fails. Arsenic is often of service. All alkalies should be given well diluted, and taken on an empty stomach before food. Then at times there is present anæmia, or the patient may have been pulled down by a severe attack of gout in some form, and then iron is indicated. But it should never be given till all active symptoms are over and the tongue quite clean, and then it should be given with alkalies and be well diluted.

As to clothing, in the pallid they must be very careful against cold, and be clad in flannel; with the florid and robust, to say nothing of the plethoric, the clothing is not so important, and these persons are usually found thinly clad.

When the pulse is tense and full, and there is risk of arterial rupture, it may be necessary to resort to the use of the lancet; ordinarily, however, smart purgation with an alkaline saline will be found sufficient to lower the high arterial tension.

Opium and mercury are, as a rule, not well borne by the sub-

jects of gouty kidneys, and should be avoided. Albumen in the urine does not positively contraindicate either drug; but in chronic renal cirrhosis, they are, as a rule, best avoided. Where there are pink lithates an occasional mercurial is often of great service. In the advanced stages these agents must be used very cautiously.

All compensatory actions are to be carefully noted, and discriminated from maladies *per se.* They are often preceded by symptoms of kidney trouble, as lumbar pain, weight over the loins, malaise, scanty urine, often approaching suppression, and the presence of albuminuria. Then comes on pretty smart action somewhere, and relief is obtained. The restoration of the action of the kidney is an important part of the treatment. The warm bath, alkaline saline purgatives, gentle potash diuretics, as the *potus imperialis,* and hot poultices over the loins (or even cupping) are indicated. If the form taken is that of diarrhœa, and it is serious, a mixture of pernitrate of iron, with a little nitrate of potash in calumba, is, perhaps, the best and least objectional form of remedial measure to adopt.

As to albuminuria, it is rarely of such a character as to call for any special treatment. Certainly cramming the patient with albuminoids is not the best practice; often to reduce them reduces the albuminuria markedly.

As to sleeplessness, it requires its own appropriate measures. Opium is not to be used if possible. Chloral, with or without bromide of potassium, is the drug for the subjects of gouty heart and kidney, with a high blood-pressure. Hyoscyamus, or tincture of hop, or lactucin, may be tried. The patient should go to bed after a light supper, a draught of alcohol, especially the somniferous malt liquors if they are well borne, and not be too heavily covered, else the body temperature will get too high. When practicable, it is well to be thoroughly tired by muscular effort.

Treatment of the Second Stage.—As the aspect of the case changes in time so must the treatment. The walls of the heart sooner or later become the seat of fatty degeneration and the dilatation sets in, and we get the evidences of heart failure superadded to those of lithiasis. Though this necrobiotic process cannot be arrested in failing hypertrophy, much may be done to retard the

downward progress of the case. Now digitalis becomes very useful, and may be given alone, or in combination with strychnia, especially if the respiration be embarrassed.

Digitalis, nux vomica, and carbonate of ammonia, form a combination very useful in the latter stages of the gouty heart, especially when there is also emphysema and chronic bronchitis. Of course it is clearly understood that this plan of treatment is only palliative; curative treatment is out of the question. Step by step the case goes down; the molecular decay of the primitive muscular fibres of the heart being followed by blood stasis, both in the pulmonic and systemic circulation. Free purgation often furnishes much relief, especially when any œdema shows itself. In fact, in some cases, the issue would seem to turn upon smart purgation. Nor does sharp purgation depress the patient at the time so much as might be supposed. The warm-bath is very useful. The reader may turn back to the case given at page 313, and peruse it at this point. Albumen begins to show itself in the urine, at first fitfully, but after a time it is persistingly present. Its presence is very ominous at this stage. There are gastric disturbances, and the sense of fulness experienced even when the stomach is empty, is added to the dyspepsia frequently experienced. The treatment of the later stage is, indeed, that of heart failure with the addition of the condition of lithiasis. The albuminoid waste imperfectly oxidized is constantly present in the blood, and sets up, when present in great excess, arteriole spasm, which may burst a rotten artery or bring the decayed heart to a standstill in diastole. The opposition offered by the arterioles ordinarily in this state gives the heart so much more to do on each systole, by keeping up the blood-pressure in the arteries. Consequently the treatment is complex, and potash must be given along with cardiac tonics, in order to cleanse the blood of its excrementitious matter. The keeping of the blood healthy, so far as this may be attained, is eminently desirable.

Basham said, in speaking of Morbus Brightii in whatever form: "The most rational and efficient principle of treatment will be that which, while not neglecting attention to the most obvious functional disturbances, nevertheless looks chiefly to the restoration of the blood to a more healthy condition, before any bene-

ficial alterations can be expected in the functions of those organs which are most obviously deranged." In some cases it is well to let the patient drink freely and then to remove the surplus water by the warm-bath, by which means the tissues of the body are well washed, and the blood cleared of its waste matters. When there are pink lithates in the water, a mercurial pill occasionally, to act upon the liver, is often of service. In order to avoid lading the blood with albuminoid waste, the dietary should not contain too much nitrogen, and beef tea should not be given in unlimited quantities nor in concentrated form, so much as made the palatable vehicle for starch in some form. The beef tea should be thickened with fine oatmeal, or biscuit powder, or baked starch or any of the numerous foods for infants now so common. Such baked starch is as serviceable for old persons with failing gouty hearts as for infants with feeble digestions. This is a matter too little attended to usually, yet it is of great importance. Then stewed fruit with cream should form a portion of the dietary. Or maraschino and cream may be given, and forms a delectable compound. Cod-liver oil is rarely well borne by these patients, but might be tried in combination with maltine. The most easily digestible foods in their most palatable forms must be furnished to the patient in small quantities at a time. Iron, though it has much to recommend it on theoretical grounds, is not well borne by gouty patients. Iron increases oxidation, and, therefore, would seem indicated, but it does not agree with such patients. The best tonic is carbonate of ammonia with a vegetable, as strychnia. It must be borne in mind, however, that both strychnia and quinine are apt to render the bladder-centres in the cord very sensitive, so that the vesical irritation caused by them often renders it necessary to suspend them. Belladonna may be tried in such cases. While the patient is still well enough to get about in a carriage, he or she may try Buxton, or Vals, or Carlsbad, or Homburg, and drink of the waters, or if the journey is too long, the salts of these waters sufficiently diluted may be drunk. The chalybeate waters of Schwalbach, Pyrmont, Spa, and St. Moritz may be useful in the middle stages, when the system has been much pulled down by a severe attack of gout, or other illness; but only after a sufficient course of alkaline waters, more espe-

cially of the purgative order. The calcic waters of Contrexville or Wildungen often give great relief, especially when there are bladder troubles, as catarrh or gravel.

When the final stages are reached, and the patient is confined to his room or house, then the lines of treatment laid down in Chapter XII must be followed; due allowance always being made for the element of lithiasis present. Indeed, the treatment of the final stages of the gouty heart will call out all the therapeutic and dietetic skill of the most accomplished practitioner; and, though cure is out of the question, much may be done to lessen the troubles of the patient's condition, and to smooth the declining path; for which both the patient and the patient's friends are grateful.

Kidney Disease the Consequence of Heart Disease.—While the subject of kidney disease being a common cause of heart disease has been very extensively investigated of late, and much information has been collected in the "Zusammenhang" of kidney and heart disease, but comparatively little attention has been bestowed on the kidney changes set up by disease of the heart, especially disease of the mitral valve, or other cause of venous congestion. Förster, Oppolzer, Traube, Rosenstein, and Von Dusch in Germany, and Sir William Jenner, Dr. Barclay, and Dr. Dickenson in this country, are the names best known in connection with this cause of renal change. In 67 cases of granular atrophy of the kidneys, Förster found no less than 26 to be accompanied by valvular disease of the heart; and came to the conclusion that valvular disease was a cause of chronic Bright's disease. In an analysis of 79 cases of valvular disease of the heart Dr. Barclay gives 28 as being also the subjects of ganular disease of the kidney. Oppolzer held valvular disease to be a cause of chronic renal disease. Traube held the view strongly, and found the bulk of urine to be lessened from the fall in arterial tension, and that this concentrated urine threw down lithates in abundance on cooling; while it was albuminous from transuded blood-serum (transudirt blutserum), and contained long hyaline exudation-casts (schlauch förmige cylinder); in fact, the results which Frerichs attained from ligature of the renal veins. Rosenstein recognized the fact that kidney disease might be in some cases the cause of

heart changes, and at other times the consequence of heart disease. In Rokitansky's school, in Vienna, the view that heart disease, valvular or other, which led to venous congestion, was a cause of interstitial nephritis, was taught. Sir William Jenner teaches that venous congestion of organs leads to a development of connective tissue in them. When in Vienna, in the winter of 1871–72, this subject attracted much of the writer's attention, and where there was heart disease of any standing the liver, spleen, and kidneys, were found to present certain alterations, viz., a development of connective tissue in them, with the certainty of a physical law. In these cases the epithelium cells were coiled up (gewundenen), and the tubules filled with blood tube-casts, or stained albuminous masses, occasionally undergoing fatty degeneration. When the kidneys were affected for long by old-standing heart disease they were small, hard, the cortical substance being small and atrophied, with the surface granular and the capsule adherent. In some old-standing disease could be clearly recognized amidst recent change from heart failure.

In Chapter IV the different changes in the viscera produced by venous congestion were described. Whenever there is fulness of the venules of a viscus persisting for some time there arises therefrom a growth of the connective-tissue corpuscles. Connective tissue, the lowliest histological form out of which the higher tissues are developed, exists in every viscus binding the higher tissues together, and forming a sort of packing in the interspaces. In the kidneys this connective tissue in health exists but sparingly; but where there has been prolonged venous congestion there is a proliferation of cells therefrom. The first effect upon the viscera is to cause them to become larger and denser, and on section a glutinous bloody fluid exudes; in time an excess of connective tissue is formed, and as this contracts, the organs become contracted, hard, and firm. The kidneys, after valvular diseases of the heart proving quickly fatal, are somewhat enlarged, vascular, deep-colored, and injected; a bloody fluid exudes on section, and to the touch they feel firmer and denser than normal; in fact, instead of a mass of vessels and tubules, with very scanty connective tissue holding them together, there is a distinct development of connective tissue. The capsule may still be smooth, but it is

either generally deep-reddish, or the surface is chequered with a stellate injection of the venules (stellulæ Verheynii), while the cortical substance is thickened, injected in streaks, or of a grayish red color; the cones are hyperæmic and darkened, while the papillæ are pale by comparison. This interstitial nephritis—for it is a parenchymatous inflammation according to Virchow—may be accompanied by metastatic nephritis from embolism, but is usually simple and uncomplicated.

When the course of the heart failure has been brief the changes in the kidney are also recent; when the heart lesion has been of long standing then the renal changes are also of a chronic character. In some cases kidneys are found which present the evidences of general recent interstitial inflammation, while they carry, as well, the evidences of old-standing disease, as pits or scars on their surface, old depressions, indicating primary disease in the kidneys, which has led to secondary changes in the heart; then the heart has begun to fail, and with heart failure comes renal change, and the implantation of recent changes amidst the old-standing disease. Consequently, when in cases of valvular disease of the heart albumen appears in the urine, and exudation tube-casts are found, the import is only too unmistakable that interstitial nephritis is being set up.

Dickenson made post-mortem examinations of 153 persons with valvular disease, of whom he found 29 to have kidneys hard, congested, and increased in bulk, but still smooth. In 67 the kidneys had granular surfaces, with more or less contracted cortices. The kidneys in cases of chronic heart disease, especially of the mitral valve, are found in two conditions. They may be hard, red, and full of blood, with their capsules abnormally adherent, while their bulk is increased, and their surface still smooth, where the congestion has been long enough to produce a general increase in the fibrous tissue; but which has not been long enough to admit of the subsequent contraction. Where the time has been long enough the new material contracts, and the surface becomes uneven and granular, and cysts are developed. As to the duration of the disease of the heart and that of the kidneys, he says, "The heart has the lead, and usually keeps it; progressive and fatal as it is, it is apt to terminate while yet the renal disorder is in a com-

paratively early stage." From this it is clear that the absence or oncome of secondary renal disease may constitute an important factor in estimating the condition of a diseased mitral valve, and furnish valuable information as to whether the valvulitis be static or progressive.

As to the treatment of this form of interstitial nephritis from venous congestion, it is obvious that all that can be done is to maintain the action of the heart, and keep up the blood-pressure in the arteries, by the administration of digitalis, and the other measures given in Chapter XII, as purgatives and diaphoretics. The kidney disease depends on venous fulness; and, if that can be relieved, the kidney changes will not progress.

This chapter is a somewhat lengthy one, but the immense importance of the subject-matter must excuse that. So much of heart disease, both simple hypertrophy and hypertrophy complicated by the appearance of valvulitis, aortic or mitral, is associated with renal changes and the condition known as lithiasis, that the subject has become one of paramount importance. So much, too, is this long-drawn-out chain of pathological changes influenced by an appropriate dietary and suitable remedial measures, that it is absolutely necessary to depict the different departures from health, as to how they have come and whither they tend, with what fidelity the writer may possess. Imperfect knowledge may render the attempted delineation less faithful than could be desired, and of this no one can be more acutely conscious than the writer.

CHAPTER XVI.

DISEASES OF THE GREAT VESSELS NEAR THE HEART—THE ATHEROMATOUS PROCESS—AORTIC DILATATION—ANEURISM.

Aneurism of the arch of the aorta and aortic dilatation are so closely allied in seat and symptoms to disease of the heart, that they must be considered in any work on cardiac diseases. Atheroma of the arterial system is a very common concomitant of disease of the heart. We saw in the last chapter how commonly atheroma is found along with the ventricular hypertrophy of the gouty heart; whether valvular disease—and especially aortic valvulitis—be present or not. Atheroma is the term used chiefly in England for the arterial changes called "endarteritis deformans" elsewhere. The disease is not due to any acute inflammatory action in the arterial coats, but is one of the interstitial parenchymatous inflammations of Virchow; and consists of a proliferation of connective-tissue elements beneath the tunica intima. The first appearance of atheroma consists in localized patches of these elements forming a papule under the tunica intima. In some cases the aorta and large arteries look as if innumerable small ricelike bodies were situated under the internal serous coat. At other times, the atheroma is found in patches, sometimes forming a ring round the artery, especially at points of great tension. The outside curve of the aortic arch is often the seat of atheromatous patches. At other times they are found at the root of the aorta, which is subjected to the full force of the arterial recoil. The root of the aorta is especially affected in aortic regurgitation, where the aorta is suddenly and powerfully distended by the contraction of the large left ventricle. The new growth itself consists of masses of young connective tissue with small, round, and spindle-shaped cells; and where there are localized patches mucin is also found, making the mass look like a piece of firm mucus. When the affection is diffuse the small

bodies are firm, solid, and resistant on pressure. "When such processes have lasted long in elderly people, and spread very extensively throughout the aorta—their chief seat—then the semi-cartilaginous thickenings are found to have undergone retrograde changes, softening them to yellowish pulpy patches, or hardening them to stone; meanwhile, the artery is wide and irregular, from partial yielding of weakened spots, so that its interior may be greatly defaced" (Wilks and Moxon).

This chronic inflammatory process becomes modified in two diverse directions during its progress, each a degeneration; (1) calcification; and (2) fatty degeneration. Calcification, or petrifaction, are more correct terms for this change than the earlier term ossification. It is induced by the deposit of lime salts in the atheromatous patches. Sometimes the mass, especially at the aortic root, resembles crumbling masses of old mortar; or the lime salts may be deposited in the chronically inflamed aortic valves and give to them a stony feel. Masses may form at the coronary orifices and occlude one or other of them. At other times the patch on the arterial wall may be infiltrated with lime salts, till it forms a plate of bonelike character and consistence. "If you take one of these hard plates from the aorta, and grind it down so that it will form a fit object for the microscope, you will see no lacunæ or other constituents of bone, but merely a number of round masses, disposed in a matrix of fibrous tissue" (Wilks and Moxon). One or more of these plates may become loose and be washed into the circulation. When the calcification is more general the arteries may become converted into rigid brittle tubes, often so impairing the circulation as to lead to gangrene, especially in the lower extremities. Such a condition of the arteries is very interesting to surgeons when about to perform an operation; such arteries being apt to break down under the ligature.

Or the mass of atheroma may undergo fatty degeneration, especially in broken-down constitutions, and where there has been alcoholic excess. Fat-granules are developed in the atheromatous patches, which become softened and yellow; cholesterin scales, and morsels of connective tissue, are found together with these fat-granules, or even drops of oil, the whole forming what is

termed the purée of pease. This softened mass may be washed into the circulation and form embolisms here and there; or even be lost in the general circulation. This mass has usually eroded the arterial wall beneath it to some extent, and leaves an ulcer in the artery (Usur). The blood finds its way through this ulcer into the arterial coats, and so forms a dissecting aneurism; or the weakened spot yields, forming a false aneurism. These aneurisms are liable to burst, leading to very sudden death.

Atheroma is found at points of flexion, as the axilla, the groin, and the popliteal space. Mr. Barker thinks he has seen it where an artery strikes against a bony ridge or a prominence, as in the pelvis. In this case the disease is very localized, and consequently John Hunter was led to tie the artery for aneurism at a point some distance above the seat of the disease.

When atheroma is general the renal and coronary arteries are apt to be more diseased than the rest of the arterial system.

As to the cause of atheroma, it is now recognized to be due to overstrain. This view was taught by Dittrich, Kirkes, and Traube, but the revelations made by the sphygmograph have put the matter beyond doubt. The general high blood-pressure in the arteries in the cardio-vascular changes described in the preceding chapter, is the great cause of atheromatous change in the arteries.

This change is set up earlier in some families than in others; while in some old stalwart families of the north of England, hardening and thickening of the arterial coats seems almost a normal senile change when old age is reached. Besides an hereditary disposition to take on this senile change prematurely in some families, there are other and malign influences which may accelerate this morbid process. Gout, syphilis, and chronic alcoholism cause the morbid process to proceed rapidly and to take on degenerative changes. Still, nevertheless, strain is the exciting cause of atheroma. If any proof were wanting to establish this, it would be readily found in the fact that so long as the mitral valve is healthy the pulmonic circulation is free from atheroma—except where there is great obstruction from disease of the lungs; but when a leak in the mitral valve throws the weight of the systemic circulation upon the right heart, then extensive atheroma is found in the pulmonary artery and its large branches. The

same changes may be set up in mitral stenosis; the obstruction at the mitral valve raising the blood-pressure in the pulmonic circulation. At the same time atheroma of the systemic circulation is prevented by mitral stenosis. Atheroma is more common amongst men, who are not only more subject to lithiasis than women, but as a rule, work harder. Very hard-working men are subject to atheroma; and aneurism is comparatively rare amongst women, and when found is usually associated with laborious occupations. Wilks and Moxon think the cerebral, splenic, and cardiac arteries especially liable to atheromatous change. "The *brain*, by means of its *superior cerebral veins*, discharges its blood forwards into the longitudinal sinus, in a direction opposed to the current in the sinus, which runs backwards; this must make the escape from the brain of the blood sent into it to be difficult, and thus lead to resistance to the flow from the arteries and tension within them, spoiling their nutrition by irritation, and so leading to 'atheroma,' and at last choking or bursting them when the chronic impediment has lasted years enough." There is no doubt about the frequent advanced change in the cerebral arteries, which are often rigid and quill-like, when the rest of the arterial system is comparatively little affected, except the coronary arteries. The *heart* is very peculiar in its parietal circulation, seeing that the ventricle's contraction throws blood into the *coronary arteries*, while it hardens and compresses the tissue of the heart, so as to impede at the same moment the passage of blood on through the capillaries, thus producing tension in the artery from the resisted current. The coronary arteries also are placed at the root of the aortic column and so are subject to the full force of the aortic recoil, unbroken by intermediate arteries. The *spleen's* circulation shows remarkable peculiarities, for great venous spaces are constructed in it, which in a sense are obliged to wait the pleasure of the liver that they may pass on their current. This tension will strain the artery and lead naturally, in the view we are advocating, to atheromatous disease of its vessels, to which it is very liable. The lower limbs, where the blood-current has to lift the blood-column in the veins, are more apt to have their vessels becoming atheromatous than are those of the upper extremities. Atheroma is very common in aortic regurgi-

tation, where the arteries are rapidly and excessively dilated by the contraction of an enlarged and thickened left ventricle. In fœtal life, when the right side of the heart sustains the weight of the circulation, valvulitis (a consequence of strain), which is pathologically allied to atheroma, is commonly found on the right side of the heart. (Strain in its Relations to the Circulatory Organ, "British Medical Journal," March 13, 1873.)

Aortic Dilatation.—This affection is commonly found with aortic regurgitation, where the aorta is acutely distended by the huge mass of blood thrown suddenly into it by the enlarged left ventricle. Its surface is roughened, and its elasticity impaired. The artery is steadily enlarged by the abnormal distending force, and the extending degeneration of its coats, until it is widely dilated.

This affection is rarely of a demonstrative character, and is often detected by the physician, when no subjective sensations have attracted the patient's attention. There is a decided increase in percussion dulness across the aorta at the second right costo-sternal articulation; there is also a harsh systolic murmur, especially when there is no aortic regurgitation. There is commonly, too, a delay in the pulse of the extremities. The most certain sign, however, is to insert the forefinger in the sternal notch, at the same time directing the patient to thrust his head forward; the heave of the aorta then becomes distinctly perceptible, and a thrill is felt. "Dilatation of the aorta, carried to a certain point, and involving the orifice of the vessel, if unattended with proportionate expansion of the valves, will give rise to patency and a murmur of reflux, which, if the degree of inadequacy be extreme, will be prediastolic in rhythm. If the internal surface of the vessel be rough from atheromatous change, a systolic murmur, located above the base of the heart, and not transmitted into the arteries of the neck, will be the result. I cannot admit that equable and moderate dilatation of the aorta is cognizable from physical evidence in the absence of the condition and attendant signs just specified." (Hayden.)

This condition is usually accompanied by, or rather preceded by, cardiac hypertrophy; but sooner or later the impaired aortic recoil leads to imperfect coronary circulation, and to degeneration of the heart-walls. It is commonly found with aortic valvulitis.

The prognosis of course is very bad; and the condition has no ameliorative treatment, except to keep the blood-pressure in the arteries low.

Aneurism.—"A man is no younger than his arteries" is the pregnant thoughtful remark of one of our greatest pathologists, S. Wilks. However well and hale a man may be in other respects, his life is to be measured by the condition of his arteries; rupture of an artery or aneurism may blot out his chances of existence with fearful rapidity. An aneurism may be of different forms. An annular aneurism may arise, according to Rokitansky, from local vasomotor paralysis. At times it is fusiform or spindle-shaped. Then there are sacculated aneurisms. These are protrusions at certain points of the artery, and may take their origin either in a weakened portion of the arterial wall, or in an atheromatous ulcer. In the latter case a dissecting aneurism may be formed from the blood burrowing along amidst the different layers of the aortic walls. This last form of aneurism may point and burst at a distance from the seat of the ulcer from which it first sprung. Aneurisms may arise from a crack in the aortic wall from a blow on the chest during the arterial systole; especially is this liable to happen when the aorta is rendered brittle by commencing general atheroma, and is mostly found among men who are the subjects of lithiasis. An aneurism once formed may burrow in various directions, producing certain subjective phenomena. "As long as the aneurism remains inclosed within the thorax, without touching its wall, diagnosis is not assisted by physical examination." (Niemeyer.) The symptoms of aneurism are chiefly due to pressure. "The pain of aneurism," says Hayden, "is mainly the result of eccentric pressure upon adjacent sentient nerves, and is referred to the seat of their distribution. It is usually shooting, paroxysmal, and of extreme severity, not unfrequently eliciting cries of agony from the unhappy sufferer; it is likewise intermittent. When the aneurism produces erosion of the osseous walls of the thoracic parietes, the pain is of a dull, aching character, accompanied at times by severe darting pains along the intercostal nerves. The intrinsic pain of thoracic aneurism, though not urgent, is not the least significant of its symptoms. It is localized, deepseated and constant, oppressive and

constrictive rather than sharp, and readily aggravated by causes which quicken the circulation, and is then associated with dyspnœa in greater or less degree. This pain is caused by tension and subacute inflammation of the sac, and by pressure upon its nerves of supply; hence the alleviation which follows the abstraction of blood or other measures calculated to reduce intravascular pressure."

Pressure upon the recurrent laryngeal nerve gives rise to loud ringing, metallic cough; and to attacks of dyspnœa due to spasm of the larynx, which may last for hours. Or there may be paralysis with aphonia, or a husky voice or stridor. These symptoms generally indicate an aneurism of the anterior surface of the ascending or transverse portion of the arch of the aorta. At other times there is pressure upon the trachea or œsophagus, producing dyspnœa or dysphagia. Pressure upon the bronchial vessels and nerves may lead to a low form of pneumonia readily passing into gangrene. Hayden thinks dysphagia from spasm is more commonly provoked by dry rather than by moist food. When the cardiac nerves are pressed upon anginose paroxysms are set up. Pressure upon the thoracic duct by aneurism is very rare. When the vertebræ are being eroded the pain is of a boring character, and increased at night. Paralysis from pressure on the spinal cord has been produced by aneurism.

There are vasomotor effects of diagnostic value. There is alteration of the size of the pupils, the pupil of the side in which the aneurism exists being contracted. The sight is not impaired, and the pupil is still sensible to light, but not to the same extent as the unaffected pupil. Bilateral aneurism may give rise to contraction of the pupil on both sides. The temperature of the affected side may be modified by an aneurism.

Inequality of the radial pulses is indicative of pressure by aneurism, or other growth within the thorax. Occasionally one pulse is almost or completely obliterated; when the aneurism involves the ascending aorta and commencement of the transverse portion, the right subclavian is involved and the right radial pulse is diminished; the left radial artery is affected by aneurism of the left portion of the arch and the descending aorta. Postponement of the radial pulse by an aneurism has been noted. When the caro-

tids are involved in an aneurism, the brain may suffer to the extent of hemiplegia. The sphygmograph furnishes evidence of aneurism, as the reader will see by referring to pages 87–89.

The progress of an aneurism of the aorta is almost invariably from bad to worse. Rupture of the sac by a blow may anticipate erosion of its coats. Dyspnœa may prove fatal, or the patient may die of inanition from dysphagia. Erosion of the spine may lead to spinal meningitis. Erosion of the sternum may lead to death from hæmorrhage. We know little of the consequences of pressure on the thoracic duct. General debility may prove fatal. Erosion of the bronchial wall may lead to hæmoptysis, and in the case of the famous surgeon, Liston, a clot formed at the orifice and retarded the final hæmorrhage for some time. Then the aneurism may burst into the veins, or arteries, or into the chambers of the heart itself. Of Dr. Peacock's collected cases three-sevenths of the whole number burst into the pulmonary artery.

The physical signs of thoracic aneurism only manifest themselves when the tumor has reached the thoracic wall; diminished respiratory murmur, or altered bronchial sounds, are too indefinite to cause more than a suspicion of aneurism. When the tumor reaches the chest-wall, then it gives an impulse which may be seen by the eye and felt by the hand. The impulse is synchronous with the cardiac beat, or follows it closely; when the tumor is in the ascending aorta, impulse is on the right of the sternum, at the second right intercostal space. When on the descending aorta, it is seen on the left of the sternum; when the transverse portion of the arch is the seat of the disease, the impulse is seen and felt at the manubrium sterni. To percussion there is dulness and a sensation of increased resistance. There is also a thrill to be felt very commonly. On auscultation, the heart is usually found hypertrophied, and the aortic second sound is accentuated, and, according to the late Warburton Begbie, may possess a "booming" character. Then there is a murmur produced by the eddying of the blood in the aneurismal sac and vibration of the aneurismal wall. When the aortic valves are healthy a diastolic murmur above the aneurism may be heard, and not the normal diastolic sound. Then there is an aneurismal sound which is not a murmur.

The diagnosis of aneurism depends on the presence of the signs and symptoms just given. When the indications are on the right side chiefly, aneurism involving the innominate must be suspected. Thoracic tumors assimilate aneurism. The following points seem to distinguish betwixt them. Thoracic cancer is usually preceded by cancer elsewhere. The pulsation of tumors is never lateral. A systolic murmur may be heard with a tumor from pressure, but there is no double sound or double murmur as in aneurism. A tumor produces little or no effect upon the radial pulses. The symptoms of aneurism vary according to the state of the circulation, those of tumor do not. There is no effect upon the pupil, nor is distant vasomotor disturbance set up by a tumor.

Dr. George Balfour relates a case where a trifling malformation of the aorta was mistaken for an aneurism. He also follows Naunyn in thinking that a visible pulsation at the second left intercostal space with a murmur may arise from mitral reflux into the auricular appendix.

Further, Dr. Peacock says: "The patients who are the subjects of aneurism of the ascending portions of the aorta are most commonly of the male sex, and at or about the middle or more advanced period of life. Not unfrequently they have been the subjects of rheumatism, and have been addicted to spirit-drinking and habits of intemperance. Most usually they present the common cardiac symptoms, dyspnœa, palpitation, and tumultuous action of the heart, dropsical symptoms, and signs of engorgement of the lungs, and parenchymatous viscera. These symptoms sometimes commence insidiously, and advance gradually; in other cases they occur somewhat suddenly, and as the result of some injury or strain." The diagnosis of thoracic aneurism, so long as it is deepseated in the thorax, is to be founded on the indications furnished by each case, not on any rules which might be laid down.

As to the prognosis of thoracic aneurism, it is very bad. Recovery is very rarely attained, and even such improvement as has been found in the case mentioned at page 383 is unfortunately not at all common. Life may be secured by care for years in cases where no improvement can be looked for.

The treatment of thoracic aneurisms consists in the first place of

avoiding all matters which would tend to increase the blood-pressure within the sac. Any increase of the internal pressure would tend to enlarge the sac, or even to rupture it. Effort especially must be avoided; no straining at stool should take place; if the bowels do not move easily the attempt to empty them should be given up, and the act deferred until the bowels will move easily, or some laxative medicine should be taken. Then there is the avoidance of all increase in arterial tension by the dietary insisted upon in the last preceding chapter.

The treatment of aneurism is the opposite of that of heart disease, except the early stages of the gouty heart. It consists in "levelling down" the patient generally to an equilibrium with his arteries. If the patient's habits are those of an invalid his prospects of life are better. If his habits are active, or he must toil, then the prospect is dark indeed.

The old starvation plan of treating aneurisms advocated by Albertini and Valsalva, is not now much in vogue, but the correctness of the principle must be admitted by all. The use of iodide of potassium, in full doses, has many advocates, especially George Balfour, of Edinburgh. A limited dietary, with absolute rest in bed, has been tried with considerable success by the Dublin School, and especially by Mr. Tufnell. The patient is strictly confined to bed, and arrangements made for the bowels and bladder being emptied without disturbance of the body; the patient only being allowed to turn from one side to the other, and that slowly and carefully. Sedatives, especially opium, are exhibited along with laxatives. Chloral, from its lowering the blood-pressure so markedly, is indicated as part of this "absolute rest" treatment. In severe pain, the hypodermic injection of morphia may be resorted to. The use of the galvano-cautery to coagulate the blood in the sac is on its trial. When the aneurism has eroded the chest-walls, then external measures are useful. To relieve the pain and tension within the sac, the Germans apply pounded ice and salt in an india-rubber bag. Lotions of lead and opium have been used with the same intent. When fairly through the bony framework of the chest it is necessary to shield the tumor from external violence. A cover of sheet lead or of leather may be moulded over the tumor and worn constantly. Such protrud-

ing aneurisms have been at times, chiefly by irregular practitioners, attacked with the lancet in mistake for an abscess; in some cases the fatal result did not follow immediately. Even when the skin is ulcerated through, a clot may form and plug the orifice. In Liston's case, where the aneurism opened into a bronchus, a clot getting into the orifice prolonged his life for some time.

The veins near the heart are rarely themselves affected, but they are not uncommonly pressed upon by tumors, either simple, malignant, or aneurismal. Sometimes the pressure is greater upon one vena cava than the other; when the superior vena cava is alone pressed upon, the resultant dropsy is confined to the head and upper extremities.

There are several subjects which it would be interesting to discuss here, but which must be omitted, however; they are aneurism of the pulmonary artery, and the effects of chronic alcoholism, and of syphilis upon the arteries, the latter having been studied by Heubner, Friedlander, and Batty Tuke, with very interesting results.

CHAPTER XVII.

MALFORMATIONS OF THE HEART.

This chapter will be a very short one, as it must be either a brief *résumé* of the subject, or a lengthy inquiry into it. No half-way account is of any value. The heart is liable to various forms of malformation, some of which interfere seriously with its functional power, while others, as a mere perforation of the interventricular septum, according to Niemeyer, are undiscoverable in life, giving no evidence of their existence. Cyanosis, or blue discoloration of the features, usually accompanied by clubbing of the finger ends, was once regarded as the evidence of congenital malformation of the heart. Congenital malformation may, however, occur without cyanosis or clubbing. Some time ago I was consulted about two brothers; the elder was pallid and reptilian, he could not walk upstairs, and when he was asked to repeat a little piece of poetry he had to be placed in the horizontal posture, in order that enough of blood might circulate in his brain to enable it to be functionally active. The younger was an active sharp energetic boy, but very blue. In the first the murmur was soft and slow; in the latter, loud and pronounced.

Dr. Peacock has made a special study of the malformations of the heart. He classes them thus:

1. Malformations consisting in arrest of development occurring at an early period of life. Heart consisting of two cavities. Heart consisting of three cavities.

2. Malformations consisting in arrest of development occurring at a more advanced period of fetal life. Heart consisting of four cavities, one or both of the septa imperfect. Pulmonary artery and aorta more or less completely developed. Defect in the interventricular septum. Constriction or obliteration of orifices. Misplacement of the primary vessels.

3. Malformations occurring during the latter period of fetal

life. 1. Defects which prevent the heart undergoing the changes which should ensue after birth. Premature closure of the fetal passages. Permanent patency of the fetal passages. 2. Defects which do not interfere with the functions of the heart at the time of birth, but may lay the foundation of disease in after-life. Irregularities of the valves. Disproportion in the capacity of the cavities, orifices, and vessels, and defects in the size and form of the heart.

Imperfect evolution of the heart is not at all uncommon. The heart even may be entirely wanting. The heart may remain of the batrachian type, and consist of only one ventricular chamber with two ventricles, when the absence of the interventricular septum is complete. At other times there is merely a slit in the interventricular septum, discovered on post-mortem examination. At times the arterial stem remains uncleft, and the aorta and pulmonary artery remain one. At other times the stem is cleft, but the relative position of the aorta and pulmonary artery is transposed.

The foramen ovale may remain patent; and where there is tricuspid stenosis it rarely closes. The Ductus Botalli may remain, and a communication betwixt the right ventricle and descending aorta may exist permanently.

During fetal life the heart is liable to endocarditis, and various malformations result therefrom. The most common results of fetal endocarditis are stenosis of the ostia, chiefly of the right side of the heart, which is the one on which most work falls in intra-uterine existence. (When a leak in the mitral valve once more throws the brunt of the systemic circulation, to a great extent, on the right heart, then right-side endocarditis once more appears. The same result is brought about if from lung disease the flow through the pulmonic circulation is obstructed.)

The stenosis is situated rather at the conus arteriosus than at the aortic or pulmonary valves. In the auriculo-ventricular ostia it is the ring round the ostium which is affected.

Stenosis may affect the bloodvessels, and in the Pathological Museum of Vienna there are, or were in 1871, two instances; one of aortic stenosis below (immediately) the Ductus Botalli; and one of stenosis of the lower vena cava, close to the heart.

In both cases large communicating vessels were found in connection with the internal mammary vessels and those of the dorsal muscles.

It has been stated that defect in the interventricular septum may be the result of a syphilitic gumma which existed there; and the part thus weakened, has given way and opened the two ventricles into one.

Then there are mere anomalies, as four semilunar valves instead of three, or, may be, two only, two cusps being thrown into one, or there may be three mitral flaps, etc. Dr. Peacock thinks that these valvular malformations predispose the valvulitis in after-life. The most interesting anomaly is, however, that of transposition of the viscera. Here the heart and spleen are on the right side, and the liver on the left. The intestinal arrangements are also transposed; the cardiac orifice of the stomach points to the right and the pylorus to the left. The lungs are transposed also. There is no history, in the various specimens to be seen in many museums, as to whether, in these cases, the individuals were left-handed in life or not.

Some of these malformations are incompatible with an individual extrauterine existence, while others are only discovered on the post-mortem table. The practical question which arises, when congenital malformation is diagnosed or gravely suspected, is this: "How long will the patient live?" As a matter of fact, such patients do not live long. As children they are usually blue, with dark lips, and are easily put out of breath. So far as I am aware they are not particularly subject to palpitation. They are usually of a lower body-temperature than normal, and are readily affected by low external temperatures. The peripheral portions of the body are affected, the lips and nose are blue; while the finger-ends are clubbed in a manner closely resembling the clubbing of chronic phthisis. Such imperfect beings have a distinctly reptilian character. They live on till puberty, which is rarely accomplished in these cold-blooded creatures; they seem to be too reptilian to take on puberty with its accompanying passions. They lead a sort of batrachian existence, a very innocent life; their mental manifestations are feeble, and the mind and body

remain childish, even when the growth exceeds that of childhood.

In a case which occurred in the practice of my friend Dr. Elliott, of Carlisle (see the "Proceedings of the Medico-Chirurgical Society," 1868), the individual had reached the stature of manhood, and was being educated for a missionary, when he died. In this case the cyanotic signs first showed themselves when three months old; yet he lived till the age of nineteen years and eight months. His heart was univentricular and batrachian, the septum ventriculorum being totally wanting. The subjects of congenital cyanosis are liable to suffer from venous congestion, and the usual results thereof, viz., development of connective tissue in the different viscera. Death may follow directly from the visceral changes, or from intercurrent disease which these imperfect beings cannot successfully withstand.

"Mere imperfection of the septum does not cause cyanosis, but is a harmless anomaly which gives no evidence of its existence during life" (Niemeyer).

As to the diagnosis of congenital malformation of the heart, it is not so difficult to make out that there is a malformation, but it is extremely difficult, probably impossible usually, to make out what the malformation is. In a case of imperfection of the interventricular septum, the result of congenital deficiency of the operculum at the foramen ovale, Dr. Mayne found a loud *bruit de soufflet* accompanying the first sound, and localized at the sternal end of the left fourth costal cartilage. The pulse was regular, soft, small, and ranging from eighty to ninety per minute. The patient was lethargic, and slight exertion quickened the pulse and respiration (Hayden). Dr. J. W. Ogle made an extensive series of observations on the patency of the foramen ovale. In only one case was a murmur heard. It was diastolic in time, and was due to coexistent inadequacy of the aortic valves. Hayden records a case where a faint systolic murmur was audible over the left apex in a case of cyanosis, with patency of the foramen ovale. Another case, with widely patent foramen ovale, had no murmur. Dr. Peacock writes, "If this be the case (pulmonary stenosis) a loud systolic murmur will be heard in the præcordial region, and most intensely at the level of

the nipple, and between that body and the sternum. It will be audible, very distinctly, in the course of the pulmonary artery, or from the base of the heart towards the middle of the left clavicle, and less distinctly in the course of the aorta, or at the upper part and right side of the sternum. If the pulmonic orifice be permanently open, as is often the case, especially where the whole of the valves are united, there may also be a diastolic murmur; but from the very small size of the aperture, in most instances, the regurgitant current is, probably, generally too slight to generate a distinct murmur. The impulse of the heart is usually powerful, and frequently a distinct purring tremor may be felt over the situation of the pulmonic orifice. The pulse is generally quick, small, and weak. If the evidence of obstruction at the pulmonic orifice be tolerably conclusive, we may safely infer there is either a deficiency in the septum of the ventricles, or a patent foramen ovale; for one or other of these defects almost invariably coexist with that condition. An aperture in the septum of the ventricles, without other malformation, would probably be attended by a murmur, caused by the flow of blood through the abnormal opening from the left ventricle into the right ventricle or auricle. The detection, therefore, of a systolic murmur at the base of the heart without signs of obstruction at the aortic or pulmonic orifice might lead to a suspicion that such a communication existed. This surmise would be strengthened if the murmur were not propagated in the course of the pulmonary artery or aorta. I do not know that there are any means of detecting, with tolerable certainty, the open state of the foramen ovale." Such are excerpts from Dr. Peacock's section on the diagnosis of congenital malformations, and his guarded expressions show how difficult a subject this has been to investigate, for Dr. Peacock's acuteness and accuracy of observation are as remarkable as his patient industry and his precision in his writings. Readers who wish to know more of the malformations of the heart must consult Dr. Peacock's classical work. My own observations extend no further on this matter, than that where there are cyanosis and other evidences of cardiac malformation present, at times a murmur can be heard which is basal in locality, and systolic in time, not very loud in character, and which does

not correspond with any of the systolic murmurs with which I am familiar.

As to the prognosis it is very bad, except in mere transposition of the viscera. A necessarily short life is surrounded by more than ordinary risks from intercurrent disease.

As to treatment, it can only be palliative. I have never seen any good come from the use of digitalis in congenital disease; though the *a priori* view that it would be useful in pulmonary stenosis seems reasonable. Rest and quiet certainly give relief; and one thing is palpable, these human reptiles must ever and always be warmly clad and protected against atmospheric variations of temperature.

CHAPTER XVIII.

THE ELEMENTS OF PROGNOSIS IN HEART DISEASE.

THE difficulties encountered in attempting to estimate the prognosis in many cases of heart disease are well known enough; how great then are the difficulties, in the way of attempting to lay down some rules for the formation of prognosis in heart disease generally, can readily be conjectured. It is necessary, however, that such attempt be essayed, though in the present state of our knowledge it is confessedly difficult; and it is eminently probable that in time our views on several subjects will be modified; that what is now said on some subjects with diffidence will, in a few years, be proclaimed from the housetops with confidence. Our knowledge of diseases of the heart is at present far from static, it is progressing, and in doing so changing materially on some very important points. The hopelessness which was once attached to the presence of organic disease of the heart, fortunately no longer obtains. The discovery of a murmur is not now regarded as the infallible indication of unavoidable and early death. We have learned much and unlearned something about cardiac murmurs and their indications. We have become sufficiently familiar with the diseases of the heart to recognize the numerous forms of disease to which that organ is subject; to discriminate one from another, to distinguish betwixt what are but conditions of passing debility from that significant and grave disease—fatty degeneration of its walls. We now know something of the natural history of the fatty degeneration of failing hypertrophy, the most common form of this necrobiotic change. Knowing something of this we can discriminate betwixt fatty degeneration in many cases and conditions which similate it closely. We now recognize the importance of the condition of the arterial walls in estimating the exact condition of the walls of the heart. If we find evidences of waning power in the heart with advanced atheroma of the arteries, we know, with much certainty, that the end is at

hand, and that all our remedial agents are powerless to arrest the swift progress to the grave. On the other hand we have learned, through how many painful mistakes, how much human agony and misery unnecessarily inflicted can never be calculated, that even a murmur, indicative of organic change in the valves, may not mean death for long years to come; that there are static as well as progressive forms of cardiac valvulitis which are not incompatible with fair length of days. Loud murmurs may exist without any evidence of impaired power, and after many years cease to be a source of alarm to the individuals, who, when the discovery of the murmur was first made, was informed he had not long to live. Of old, when a murmur was heard, it was at once assumed

> "It is the rift within the lute
> Which soon shall make the music mute."

But now we know better. We now recognize the fact that it is often much easier to detect a murmur than to appraise its significance, and to understand its interpretation. We are not now so hopeless about the prognosis of cases where murmurs are heard; in the first place we search out the characters of the murmur, ascertain if it is not hæmic or dynamic. Having decided that it is organic, we have next to proceed to determine that it is not a congenital condition of a valve, before we assume that it is the result of disease; and even when at last we feel convinced that it is the result of disease, there are yet points to be settled as to whether the mischief is non-progressive or not. Should the evidence unfortunately point to progressive valvulitis, still there is the estimation of the patient's power of compensatory muscular growth, and the effects of diminished demand upon the heart by absolute rest, to be determined; and finally, we have to deal with the question of remedial measures—of which last subject we know much more now than we did even in the decade immediately preceding this. Indeed, there is much to encourage us in reference to heart disease; we now clearly recognize the treatment of acute valvulitis, how by absolute rest at the time, and for some time after the inflammatory storm has passed over, we can limit the valvulitis, and with it the consequential deformity; we now understand the effects

of a long maintained high blood-pressure upon the heart—alike its walls and its valves; and can comprehend with much distinctness the effects of a proper dietary upon the arterial tension, and, with that, possess definite impressions as to how we can retard the progress of the gouty heart, and prolong the patient's existence.

Much, indeed, has been learned of late years which tends to disabuse the practitioner of the present day of views, formed in all honesty but from imperfect data, held by our predecessors about heart disease; physiology has stepped in and thrown a flood of light over subjects shrouded in darkness, from which clinical medicine alone, or even with such aid as morbid anatomy can afford, could never have rescued us, as the nature of angina pectoris for instance. The careful, steady, painstaking inquiries into the action of remedial agents has taught us much that is already invaluable about the action of agents upon the organs of the circulation, and promises more. We now understand how hypertrophy is brought about, under what circumstances dilatation is found; and consequently we can now intelligently set to work to aid the natural attempts to develop hypertrophy when compensatory hypertrophy is called for.

Much of our present knowledge is due to the recognition of the fact that there is "heart disease and heart disease." Our forefathers were satisfied with the recognition of organic disease of the heart, and when they found that some cases died suddenly shortly after the discovery was made, they reasoned that a like result would occur in another case when they discovered organic disease, and gave a bad prognosis where the case went on for years without alteration; but such conclusion they reached through their ignorance and not through their knowledge of the subject. In making such statement there is no intention to disparage the intellects, or the application of the pioneers of our investigation into the obscure domain of diseases of the heart. The names of Laennec, Corvisart, Bouillaud, Hope, Latham, C. J. B. Williams, Stokes, and others scarcely less illustrious, will remain forever famous. We must recognize how much they have done and how great is our indebtedness to them; how much order they brought out of chaos, and what a legacy they left to us; and how, in our turn, it behooves us to do something to dispel the clouds of error

and to spread the light of truth by further observation and investigation, so far as we can, in imitation of these brilliant examples.

So early as the days of the "Library of Medicine," Dr. Joy wrote: "The term 'disease of the heart' stood in times past, with many members of the medical profession, as it still does with the public, for a uniform and general affection of this organ, of an utterly intractable and necessarily fatal nature. More accurate investigation into the symptoms and progress, and still more into the morbid changes of structure, presenting themselves in the individual cases passing under this title, has eventually proved how erroneous is the above conception in all its parts."

Diseases of the heart are no longer considered of so uniformly hopeless a character as we find ascribed to them in the pages of older writers, and even of Senac and of Corvisart. An improved diagnosis, and a more correct knowledge of the varieties of these affections, enable the practitioner of the present day to discover them earlier, and treat more appropriately and vigorously than his predecessors, such as are still within the reach of art. What was said then applies even more trenchantly to the present. Treatment now is widely different from what it was then; can be more accurately applied, does more good, and, we fervently trust, less harm than it too commonly then did.

Once it was imagined that any wound or injury to the heart was at once and instantly fatal; at pages 246–7, we saw that a large percentage is recovered from.

In estimating the prognosis of any, and indeed almost all, if not all, diseases of the heart, the first point to be considered is the condition of the muscular walls. We saw in the first chapter how the heart is primitively a muscular sac; how, as evolution proceeds, valves are developed which economize the muscular efforts. When, then, in the fully developed four-chambered heart of the mammal, the valves are rendered less perfect than they normally are—the heart is degraded to a lower form, and rendered once more, more or less, a mere muscular sac. Of course this degradation can only proceed to a certain point compatibly with the existence of the injured individual; even though absolute rest is enforced, and the individual leads a life but little less

active than a mollusc. In disease of valvular origin, and also in disease where the valves are not involved, the condition of the muscular walls is all-important. The commencement of dilatation in a heart once firmly hypertrophied is ominously suggestive. Where there has existed dilatation the restoration of the heart to its normal size is of the happiest prognostic import (page 116). The loss of force in the heart's beat is bad, prognostically, especially if it persist under active treatment. The same may be said of the loss of the first sound of the heart. But it is well to bear in mind the simulative conditions which are described in Chapter IX, else an error may be easily made. Then there is the loss of rhythm. Alone this may be merely a neurosal disturbance; but, if found with the other evidences of failing power, it is very suggestive. Thus irregularity produced on slight effort is very bad from a prognostic point of view. Chronic irregularity is less ominous where palpitation is induced by effort, than where intermittency is so set up; this last indicates that the heart is unequal to the active condition of palpitation. A number of rapid small beats followed by a long pause and a strong beat indicate dilatation, not rarely mixed up with fatty degeneration. When a strong beat is felt amidst a number of small imperfect beats in simple dilatation, or mitral insufficiency, and in comparatively young subjects, not presenting the evidences of arterial degeneration—that strong beat is encouraging. It is not always possible to discriminate betwixt a heart passing through a stage of temporary inability, and an established condition of dilatation, especially if mitral disease be also present, without waiting and watching the effects of a course of appropriate treatment. The treatment will greatly, indeed, in many cases, profoundly modify the prognosis. Unsuitable treatment will often verify a hopeless prognosis, possibly not warranted by the facts of the case; and the patient drops into a grave never dug for him by nature; or in other cases drops into it years before the time he would reach it under judicious treatment.

Impairment of the general power and growing inability to undertake exertion is one of the gravest indications of advancing disease in the heart-walls. The effect of effort is usually, too, the

safest and most trustworthy guide as to whether the heart symptoms are neurosal, or due to organic changes.

If the heart's rhythm be notably disturbed by effort, or a mitral murmur inaudible on quiet is revealed by effort, then the prognosis is unfavorable; always and provided that the practitioner bear in mind Latham's second form of cardiac unsoundness, given at page 157. When the powers are notably impaired, and the gait is altered, being watchful, yet uncertain—then structural decay is almost certain.

If there are evidences of general decay—if the patient looks much beyond his or her years—then the prognosis is very bad. If there is a distinct want of proportion betwixt the physiological and the chronological age, the real age exceeding the age by the birth-register, the probability of structural disease is rendered terribly certain. The two forms of arcus senilis have a definite value (page 217). If there be also degenerate arteries meandering along the temples, the diagnosis of fatty degeneration is almost beyond dispute.

Long syncopal attacks are of bad omen; comparatively less so if there be also aortic obstruction, of which long fainting fits are almost pathognomonic. The falling off of the bulk of urine is also prognostically valuable. The bulk of urine is in proportion to the arterial tension; as long as this can be maintained the bulk of urine keeps up; but when the bulk of urine is notably lessened the prognosis is gloomy. Especially is this the case with the gouty heart, and the fall indicates that the first stage is giving way to the later stage. Venous congestion is a very significant indication that the heart is failing, and the appearance of albumen in the urine, especially if accompanied by recent exudation-casts, is of the very worst omen.

Then there is the prognostic value of cardiac murmurs of organic origin. This is a matter of very great importance, and will be found to require careful attention.

There are some wise and judicious remarks of the late Dr. Stokes which are worthy of serious and attentive thought. He says, "The doctrine that disease of the valves, when it is uncomplicated with any functional or organic lesion of the muscles of the heart, is often so latent as to be undiscoverable without phys-

ical examination, is one of the great truths for which we are indebted to Laennec. And it is not yet sufficiently insisted on that valvular disease, even to an extreme degree, may affect the heart without there being anything in the previous history or existing symptoms which could lead us to suspect the existence of such a lesion."

A slow and, as it were, silent disorganizing process may be developed in one or more of the valves of the heart, without pain, without irregularity of action, without any circumstance which could awaken the attention of either the patient or physician; and thus years may pass by, the patient fulfilling without inconvenience all the duties of an anxious, active, and energetic life.

But with the want of symptoms there is, doubtless, for a period which is undefined, absence of physical signs as well; and, though the disease is manifestly progressive, no murmur is established until the mechanical change has reached that point which is competent to produce acoustic signs attendant on the flow of blood through the altered orifice. Thus it often happens that we may, with great care, examine the heart and find no evidence of disease; yet in a short time—it may be in a few days—manifold physical signs are developed which indicate not a recent and acute disease, but an extremely slow and long-existing affection, yet one which had not, until the period of the second examination, arrived at the point when it was at last attended with acoustic phenomena.

In the chapter on carditis I have dwelt on the error which is so commonly fallen into of considering a murmur which has existed for a long period, but was then for the first time observed, as evidence of a recent and inflammatory affection. The same error is too often witnessed in the case now under consideration, and, as might be expected, the same disastrous consequences are found to follow.

The effects of injudiciously communicating to the patient that his heart is organically diseased, in conjunction with those of an ignorant and destructive medication, produce that very condition the absence of which has been the patient's chief safety. The heart becomes irritable, irregular, perhaps excited, and it is then no wonder that the *symptoms* of disease are superadded to the *signs*.

"The recent development of the signs of a chronic, long-preexisting disease is a circumstance which should be known to all who are concerned in the medical examinations for life insurance. Thus, it may happen, a life is passed as insurable after a careful examination. The insurance is effected; and yet in a short time the individual exhibits all those signs of morbus cordis which are supposed to indicate chronic disease. He may die of this disease within a few months after the completion of the insurance, and the payment of the sum insured then be contested on the ground that the disease was overlooked. I have known all the signs and symptoms of permanent patency of the aortic valves to occur within a few months after the effectuation of a large insurance, and yet at the period of the medical examination, which was made by one of the best observers in this or any other country, no signs of disease of the heart existed. In the same way I have known the signs of chronic mitral disease become most strongly developed in the course of a few days. These facts are of practical importance, for, in the case of a judicial trial, on the ground of the incompetency or neglect of the medical examiner, many professional witnesses would incline to the opinion that the affection had been overlooked rather than it had become developed in so short a time after the examination. They would be influenced by the opinion that the development of disease and of its symptoms and signs are concurrent, a doctrine which we have seen to be untenable in acute, and, of course, far more so in chronic, disease.

He then speaks of another condition. "It would appear, however, that some valvular diseases, at all events, are either not progressive, or that they advance with such extreme slowness as to constitute a class of their own of cases very different from the more common examples of these affections."

He then relates a case where a patient, who had hitherto had excellent health until an attack of influenza, consulted a physician, who found a "bellows murmur masking the first sound of the heart on the left side." He was ordered to abandon his active habits and adopt those of an invalid. He, after awhile, feeling the restraint irksome, consulted Stokes, who took into consideration his previous health and habits, and who concluded: "I suspected that the murmur was indicative of some very old, passive,

and stationary valvular disease, and this suspicion was converted into a certainty by the patient informing me that seven or eight years previously he had suffered from a severe attack of rheumatic gout, which affected many of the joints. There could hardly be a doubt that the murmur was established at that time, but that the diseased action had not been progressive; the valves had been mechanically altered, but not to such a degree as to interfere materially with their functions. So that we had in this case to deal with the cicatrix of a wound, as it were, rather than with the wound itself." He was permitted to resume his active habits without any bad result.

"That this individual has had a continued mitral murmur for upwards of twelve years there cannnot be any reasonable doubt, and the case is strongly illustrative of this principle in practice—that we are not to confound the effects of a disease with the disease itself; and again we are not rashly to change the habits of living, as to exercise and the use of stimulants, in a patient who has been the subject of a chronic local disease, if we find that under the regimen in question, local disease had not been progressive, and that the general health has remained unimpaired."

From these remarks of this accomplished observer and exact reasoner it will be at once evident how difficult it is to form a prognosis from the physical signs of many cases. There are cases where a loud mitral murmur causes no apprehensions; there are other cases where mitral disease can be suspected before a murmur is heard, announcing the fact that a certain point of disease has been reached. From an insurance point of view the diagnostic and, still more, the prognostic value of cardiac murmurs needs much further investigation. I venture to predict that when this is efficiently done our present views as to the overwhelming significance of murmurs will be profoundly modified.

Assuming, however, that valvular disease is indicated, each by its special murmur of time and locality, we may proceed to the consideration of the prognosis of the different valvular lesions of the heart.

Dr. Peacock, in a brochure on "The Prognosis in Cases of Valvular Disease of the Heart," says: "The order in which these conditions should be placed, as indicating their relative

danger, beginning with the more serious affections, would therefore be as follows:

Aortic Regurgitant Disease.
Mitral Regurgitant Disease.
Mitral Obstructive Disease.
Aortic Obstructive Disease.

"The comparative rarity of serious affections of the right side of the heart, and their being usually combined with other defects in the conformation of the organ, make it difficult to estimate the relative danger which attends them, as compared with diseases of the left valves." Dr. Peacock puts the positions correctly here, unless some doubt may exist as to the relative position of affections of the mitral valve.

But admitting the correctness of this arrangement, this rule cannot be applied so as to be made the gauge of each case of valvular disease. The tissues of some persons—and apparently indeed of some families—seem peculiarly irritable; so that valvulitis once started goes on and on from bad to worse, and no skill nor care can reduce the valvulitis to a static condition. Thus in certain families member after member die off early with cardiac valvulitis—the system making no stand, and the compensating muscular growth never being efficiently developed. Where there is such an unfortunate family history, the least departure from a condition of perfect health will cause grave anxiety. In others, again, the tissues seem of quite another order, and in some cases of aortic regurgitation, where the mischief was set up by an acute attack of valvulitis, the condition has remained static for many years. One such case in a medical man is well known to me: for twenty years and more he has led an active life, and had a large practice; has at times worked himself nearly to death's door; has come round, and is now much the same as he was when he consulted me first, several years ago. His heart "lets him down," and he is a crippled being; yet the disease at the aortic orifice manifests no tendency to grow worse; indeed, after the first mutilation of the valves the condition has been absolutely static. In an hospital patient, a woman of over forty, there has been aortic regurgitation also for over twenty years; there is now an enormous ventricle

and the most startling "locomotive pulse" I ever encountered; the pulse-wave can be seen above the elbow flashing down the radial artery, over the back of the thumb, and on to the proximal end of the forefinger, on the right hand, with marvellous distinctness.

In forming a prognosis, either in mitral or aortic disease, it is of the first importance to distinguish betwixt valvulitis due to an acute attack, whether with or without rheumatic associations, and the more gradual but steadily progressive or sclerotic or contracting valvulitis, due to strain and preceded by a certain amount of muscular hypertrophy, as in the gouty heart, for instance. The first form may remain static for years; the latter is ever, even if, in some cases, but very slowly progressive. Acute aortic valvulitis, it has appeared to me, is more common in girls than in boys, and this is a matter of moment, prognostically, as marriage acts most prejudicially on women with aortic regurgitation.

As to mitral disease in young subjects, it is certain that it is much more frequent in little girls than in little boys. It is set up by acute endocarditis, which is not always associated with acute rheumatism or scarlet fever. In many cases the mischief wrought is slight, readily admitting of muscular compensation, which can easily be maintained; in the slighter cases the troubles of puberty are surmounted with ease; but in others, where there is defective systemic power as well as valvular disease, the time is one of great trial, the pubertal changes are not successfully accomplished; instead thereof there are manifested indications of heart failure, including dropsy, and the patient sinks. I have not been able to trace any of these cases to matrimony, and so cannot say what the effect of their spousal duties are upon such cases.

In this class of cases there seems little influence exercised by the form of mitral disease, whether it be stenosis or regurgitation. In the Pathological Institute of Vienna, if my memory serves me right, mitral stenosis seemed very common, indeed the prevalent form of disease; swiftly following upon rheumatic fever; and terminating fatally a year or eighteen months only after the primitive endocarditis. Such a rapid progress, fortunately, is not common in my English experience.

The subject of static and progressive valvulitis is one which

must, in the future, attract more attention than it does at present. Latham and Stokes both recognized these two forms of valvulitis, but the matter has attracted little or no attention among more recent writers, and yet it is no unimportant matter; what are the circumstances under which acute valvulitis becomes arrested at a certain point and becomes static; what the influences which cause the disease to progress to a fatal issue; what our means and measures by which we can favorably affect the course of the valvulitis, are surely matters of primary importance.

The development of tricuspid disease, secondary to mitral disease, is of great significance, prognostically. Yet, so far, we do not know whether it is the development of secondary tricuspid disease which is the essential factor in cases of mitral valvulitis going swiftly downwards, or not. Of course the development of a tricuspid murmur, in cases of mitral disease, is an indication of the worst prognostic omen. As to the development of primary tricuspid disease I can only recall one case; it occurred after acute pneumonia in an old man of eighty-four, very active for his years; dropsy showed itself while he was still walking about his garden, and the sequelæ of tricuspid regurgitation showed themselves quickly one after another, and the case soon ran its course to a fatal termination.

As to pulmonary disease it is always congenital, and is so rare that no rules as to its prognosis can be formulated. As to the prognosis of cyanosis, *i. e.*, of cases of congenital malformation, it is given in the last chapter.

The question of how far we can have a murmur—the indication of insufficiency—without valvulitis, is a somewhat difficult one to answer. Peacock, Herbert Davies, and others, hold that dilatation of the ventricle may proceed to such a point that the ostium becomes so enlarged that the mitral vela become incompetent to close the orifice on the ventricular systole. On the other hand, Rokitansky teaches that the vela and cords stretch alongside with the muscular walls. Wilkinson King found, in his experiments of forcing water into the left ventricle, that the mitral valve but rarely could be made to leak, while the tricuspid, usually, readily leaked. Yet Oppolzer and Kürschner say that such tricuspid leakage is anatomically impossible.

Clinically, it would appear, that passing tricuspid regurgitation, under conditions of great distension of the right ventricle, is not rare; and that there are cases where there is temporary mitral insufficiency from acute dilatation of the heart, which is removed by treatment which reduces the dilatation.

Where a certain injury has been done by valvulitis—so much and no more, and that, too, static—and there is enlargement of the left ventricular chamber and a regurgitant murmur, the murmur not uncommonly passes away under treatment. A murmur is produced at times, according to Pearson Irvine, when there is a dilated left ventricle, not perfectly emptied on systole, from the impact of the incoming current from the auricle upon the fluid already in the ventricle.

As to the prognosis in hæmic murmurs it is good. In those cases where the lung does quite cover the pulmonary artery, the loud murmur, aggravated by forced expiration and lessened by deep inspiration, has no prognostic significance.

Dynamic mitral murmurs commonly disappear, in a little time, under appropriate treatment.

But in the consideration of the prognostic indications of murmurs of organic origin, there is one matter which calls for more serious attention than has hitherto been accorded to it in works on diseases of the heart; and it is this: Valvulitis is not always the commencement of the patient's troubles, nor the first departure from health in disease of the cardio-vascular system. Very frequently in advanced life aortic valvulitis—and mitral valvulitis, too, I am beginning to observe—are not primary diseases, but secondary to certain changes which have preceded them. These changes are described in Chapter XV, "The Gouty Heart," and consist of a high blood-pressure in the arteries, maintained for long betwixt an hypertrophied left ventricle and hypertrophied arterioles.

The overstrain to which the valves are subjected under these circumstances leads to a chronic valvulitis of progressive character. When such a lesion is found, its prognostic indications are very serious. The valvulitis is one of the later outcomes of a long-standing pathological process, and this adds to its gravity. The hypertrophy found under these circumstances partly precedes, is

partly consequential to the valvulitis. Here the valvulitis is progressive and not static; compensatory hypertrophy is not readily developed with an inelastic aorta and thickened coronary arteries, and the case goes steadily downwards. Of course, where the patient's means will permit of a very quiet existence being maintained, the progress is much slower than in those cases where toil is unavoidable. The association of aortic disease with the gouty heart is now generally recognized, and soon, when attention is directed to the subject, it will be seen that the mitral valve also is apt to become the subject of chronic inflammation when long subjected to overstrain by an hypertrophied ventricle. There is one point of interest connected herewith, viz., aortic valvulitis with the gouty heart usually assumes the form of stenosis, as regards its morbid anatomy, though a certain amount of regurgitation may also be present; while, in my experience, mitral stenosis is rare with elderly persons, the form found with the gouty heart being usually that of insufficiency. In those cases where hypertrophy of the left ventricle has been found along with mitral stenosis, it is impossible to escape the conviction that here the mitral valvulitis was secondary to the hypertrophied ventricle.

The association of aortic valvulitis with pre-existing muscular changes gives importance to the recognition of valvular change preceding the appearance of a murmur, the premurmuric stage of valvulitis, discussed at p. 179. The accentuation of the aortic second sound, and the occasional muffling, indicating commencing change in the semilunar cusps, possess a significance greater than is yet generally accorded to them. In all cases it is not necessary to wait for the ominous sound of a murmur to tell that mischief is afoot in the aortic valves.

In making an estimate of the probable duration of life in cases of valvular disease, the form of disease is the first factor to be considered. Aortic regurgitation is, usually, swiftly fatal, from the nutrition of the heart failing early, the blood escaping past the coronary orifices, instead of eddying in the sinuses of Valsalva; indeed, Balthazar Foster has pointed, with terrible distinctness, to the effect exercised prognostically, by the fact of which aortic cusp is ruptured—if the one behind which there is no coronary artery, then the prognosis as to duration of life is better than if

either of the other cusps are torn down. Then comes the next factor—the family history, a matter of scarcely less importance; this should be ascertained as far as possible in every case. "The apple does not fall far from the tree" holds good of physical disease as well as of mental traits. Often the prognosis formed will determine the patient's future life; on it hangs his decision about important business matters; the prognosis may be a very grave matter years before the mischief, looming in the far distance, shall become a present reality in many cases; and the material for its formation should be carefully collated before the attempt is made to calculate the patient's position and prospects of life.

The question of marriage, by those who are the subjects of chronic valvular disease, has been casually alluded to above.

Dr. Angus Macdonald has carefully studied the subject as regards women; he writes: "I shall now conclude my subject by one or two practical deductions from the views that have been maintained in the preceding pages:

"1. Chronic heart disease ought to be looked upon as a grave contraindication of marriage, more especially if it assumes the form of anything approaching to severe stenosis of the mitral, or to serious aortic incompetency; in such cases we ought, if consulted, to dissuade from marriage.

"2. There is less danger in the case of mitral insufficiency, pure and simple. But still the risk is, even then, considerable.

"3. In all cases, when consulted, we ought not to give our sanction to marriage, if, in connection with chronic heart disease, there are any serious symptoms of cardiac disturbance present, such as attacks of dyspnœa, breathlessness, palpitation on exertion, hæmoptysis, etc.; and this injunction ought to be the more imperative the younger the patient, and the more recent the acute disorder which has given rise to the chronic lesion.

"4. Such patients as are married and have chronic heart disease ought not to be allowed to suckle their children, as that appears to keep up the cardiac hypertrophy, and increase the risks likely to arise from the defective heart.

"5. All possible causes likely to produce inflammatory action in the lungs, and severe exertion should, if possible, be avoided

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during the pregnancy, and more particularly during the latter months of it.

"6. Premature labor should seldom, or never, be recommended, because it is so much more likely to do greater harm, by disturbing the action of the heart, and the condition of the lungs, than any good it might produce by terminating the evil effects of the pregnancy. It is always to be remembered that relief of symptoms is not certain after delivery, or, indeed, anything like certain.

"7. The only conditions which seem to warrant the induction of premature labor are the presence of influences which unduly distend the abdomen, and thus keep the diaphragm in a state of continuous elevation.

"8. The same general principles of management ought to guide us in the case of a patient with chronic heart disease during pregnancy and the lying-in period, as are followed by us in dealing with patients who suffer from heart disease apart from pregnancy.

"9. In almost all cases I have met with chloroform has been given, and apparently with benefit, during delivery. If carefully administered, I think it cannot but be useful in all cases.

"10. All legitimate means ought to be used to lessen the effects of the down-bearing pains, and, therefore, the judicious and timely application of forceps, or, in suitable cases, the performance of version, is extremely important if the second stage of labor happens to be in any way prolonged. In case of a large amount of liquor amnii, timely rupture of the membranes is calculated to be of great service, as it allows the diaphragm to descend, and thus lessens the embarrassment in the lesser circulation.

"11. Increased experience warrants me in believing that the mortality following pregnancy complicated by chronic disease of the heart, may be greatly lessened by due precautions during pregnancy and delivery, especially during the latter."

Such are Dr. Macdonald's conclusions from his study of this subject. ("Heart Disease during Pregnancy," etc., p. 282. J. & A. Churchill, 1878.) I should, however, point out that in section iv his views on hypertrophy of the heart are not in unison with the views put forward all through this volume.

The prognosis in neurosal affections of the heart but rarely

involves the question of life; it concerns time and persistence of the affection, as pointed out in Chapter XIV. These affections are often persistent and intractable, even to the most artfully devised plans of treatment. At other times the case takes a turn for the better readily; while the cardiac troubles of the menopause commonly disappear when that period is passed. The irritable heart often cripples, but in my experience it does not kill (p. 366). It may give the sufferer much anxiety, but it does not apparently shorten his days. Neurosal affections of the heart are persistent and intractable, but it has not yet been demonstrated that they in any way predispose to organic disease which kills. According to the view that the "worry," the wear and tear, of modern life is productive of organic disease, the irritable condition should be associated with actual disease; but this has not yet been demonstrated.

Embolism, as a result of heart disease, is a factor not to be forgotten in forming a prognosis; but as to the formation of any rules on the subject there are not yet sufficient data for this to be done. Apoplexy is a danger which is never absent in the gouty heart, even in the later stages when the heart-walls are becoming decayed; for if the heart's walls are degenerate the encephalic vessels are also in an advanced stage of atheroma. "As disease of the vessels accumulates in the course of life, so necessarily with it the tendency to apoplexy increases with age, and, *cæteris paribus*, the older the person the more liable he is to an attack; but the great predisposing cause is granular disease of the kidney. This is, indeed, so frequent an associate of apoplexy that you will be surprised at its almost constant repetition in the cases that occur in the post-mortem room. Thus of the last seventeen cases of fatal apoplexy we inspected, fourteen had granular kidneys" (Wilks and Moxon). Not uncommonly when a patient with lithiasis has his blood more than usually surcharged with waste, there is a sudden rise of blood-pressure in the arteries from arteriole spasm, and then one of the thin-walled arteries in the brain gives way; or at other times, and in other persons this sudden rise of blood-pressure brings on an attack of angina pectoris; so long as the heart-walls are sound, the danger in anginal attacks

is comparatively slight; but when they become decayed then each attack is fraught with imminent danger.

When "clubbed fingers" are found along with cardiac disease they indicate a certain chronicity which is not of bad omen; but their prognostic value is small. They are the result of venous stasis leading to a growth of connective tissue. The same growth is seen in the fulness of the lips and the alæ nasi of young subjects of valvular disease. They are also commonly seen in congenital heart disease, where the thickened finger-ends are of deep blue color, as are also the lips and cheeks. Where there is this blue coloration of the finger-ends the prospects of the case are bad.

And now we may consider some relations which exist betwixt disease of the heart and disease in other organs.

Disease of the lungs is commonly found along with disease of the heart. Affections of the respiratory organs also usually produce changes in the right side of the heart. The right side of the heart is always enlarged in chronic bronchitis with emphysema, and is a very serious complication, to be borne vividly in mind in arranging the treatment. Cirrhosis of the lung commonly leads to right-side dilatation, and the same may occur from pleuritic effusion. The anasarca, which shows itself in some cases of phthisis, is probably connected with failure of the right ventricle, and is of the worst omen.

It has been asserted that phthisis is not found with valvular disease of the heart. This fact did not escape the attention of Laennec; and Rokitansky found, as a pathologist, that chronic endocarditis, or valvular disease, is rarely or never accompanied by any evidence of tubercle, except traces of old-standing disease long preceding the heart disease. Traube thought this freedom from tubercle was due to the fact that the free transudation of blood-serum into the lung-tissues, in mitral disease, was opposed to that condition of imperfect cell nutrition which favors the growth of tubercle. On the other hand Lebert regards congenital pulmonary stenosis as a predisposing cause of phthisis. In five such cases which have come under Dr. Peacock's notice, two had uncomplicated pulmonary stenosis, and both died of phthisis; the other three had other defects in the heart, and did not have phthisis, but their lungs were found much congested. When phthisis in

young subjects is accompanied by dilatation of the heart the prospect is very bad.

Thoracic deformity is very commonly followed by changes in the heart, especially at the pubertal period.

Rokitansky pointed this out, and showed how the altered relations of the thoracic viscera and the tortuous changes in the aorta combined to obstruct the blood-flow, and so led to hypertrophy. Hunchbacked children are liable to die about the age of puberty with all the evidence of heart failure, including dropsy; and all persons so deformed are liable to heart troubles.

Investigation into the relations of heart disease to insanity has furnished but negative results. During my visits to the West Riding Asylum, in connection with the medical reports published by the accomplished ex-superintendent, Dr. Crichton Browne, I made repeated examinations of the patients; and, contrary to expectation, found that there was apparently no connection betwixt heart disease and insanity; the insane were not more liable to heart disease than the sane; nor was there any evidence forthcoming that disease of the heart predisposed to insanity.

There still remains one or two points to be reviewed ere this chapter closes, which bear on the relations of medical men to each other, as well as the prognosis of each case. Differences of opinion often apparently exist, where they do not do so in reality, from the different phraseology used by the different individuals; and patients are often sorely perplexed thereby. For instance, hypertrophy of the heart will be spoken of by one man as a disease; while another will express himself as rather glad of it than not. What is the patient to think of this discrepancy? Then again acute attacks of gout are described in a loose manner as attacks of rheumatic fever, on the papers of candidates for insurance; with the awkward result that the proposals are declined. How many cases of irritable heart have been assumed to be cases of commencing disease, and the persons made anxious and miserable unnecessarily in consequence? These apparent discrepancies are most likely to arise betwixt an elderly practitioner using an antiquated phraseology, and a consultant of the modern school. Great care, therefore, is incumbent on the latter that he take pains to explain the altered phraseology to the patient; so that he shall not do any injury to his professional brother.

At times, however, actual grave differences may arise without a lack of skill or care on either side. Murmurs come and go, are one day audible, another day not to be heard. In the case recorded in Chapter XII, pp. 313–15, this is well seen; the murmur at times cannot be detected; at other times it is readily heard. In such cases difference of opinion may easily arise. Then again in double aortic and double mitral disease, the character of the murmur varies; at one time the obstructive factor is predominant, at another time the murmur is prominently that of regurgitation. Here again honest error may be committed—for error it would be apt to be regarded in the family practitioner if a consultant happened to differ from him in opinion. Differences of opinion may then arise without error on either side. One of my patients, an intelligent clergyman, gives in most precise phraseology a lucid account of his heart where aortic regurgitation was diagnosed some twelve years ago, yet there is not now a trace of such a thing. When I mention that this diagnosis was made by Dr. Walshe, no reader will think that there was any mistake in the diagnosis then made. The only solution of such a curious circumstance is that the regurgitation was due to dilatation of the aortic root which has now been remedied. Such aortic patency Dr. Sibson regarded as the case with the late Dr. Murchison. It is equally certain that this clergyman has no aortic, or other murmur now. This capricious behavior of murmurs is most important from an insurance point of view; and needs further observation and investigation.

At times very serious and valvular disease of the left side of the heart may exist with scarcely any murmur at all, when the tricuspid orifice is affected; the blood-current coming over to the injured left side not being sufficient to evoke a murmur.

Finally, in forming a prognosis in cases of disease of the heart, it is well to bear in mind the relations of disease in the heart to that in other organs; to recognize the consequences of a lesion in the heart, as congestion of the liver or kidneys; to discriminate what are its causal relationships, as Bright's disease. Latham says truly: "Observation has traced back, with fearful fidelity, a long line of formidable and fatal diseases to their pathological parentage in the heart. But that which is constructed of preceding or

coincident events is hitherto less perfect; yet observation has been able to assign to some diseases of the heart a sure origin in, and a still continued alliance with diseases of other organs, or of the constitution at large," and further experience of the profession at large is demonstrating more and more the truth of this. The gouty heart is more commonly recognized now than hitherto, as is also the irritable heart with its neurosal relations. Uterine troubles may result from disease of the heart, while at other times they give rise to disturbance of the heart's action. Psychical alterations may be originated by changes going on in the heart; just as psychical perturbations may set up disturbed action in the heart. Dyspepsia may be a cause or a consequence of cardiac disturbance. The heart-specialist, of all specialists, is not likely to get into a groove, or to see the whole field of pathology from the standpoint of the circulatory centre only. To do so would be as unwise as to make astronomical observations from the earth as a standpoint; the astronomer makes for himself an ideal standpoint from which he can survey the universe, and thus note the earth's exact place in it, and so make his calculations accordingly; and the heart-specialist must go and do likewise, else he will commit avoidable error. Heart diseases must be looked at from their causal associations, in the interests alike of physician and patient, and not regarded solely in relation to their systemic consequences, if we wish our knowledge on the subject to grow, to blossom, and to bear fruit. One result at least will be attained by such attitude, and that is the mysteriousness which is generally thought to shroud all heart disease will be largely removed, and with that the dread and terror with which heart disease is generally regarded. Disease of the heart is produced in the same intelligible manner as is disease in other organs, and the causal relations of heart diseases are now fairly recognized, while we have learned much how to ward off heart disease as well as to give relief when disease is established. Heart disease is no longer the hopeless matter it was regarded of old, and increasing knowledge of the natural history of the different diseases of the heart is leading to excellent practical results, as to prevention as well as to alleviation.

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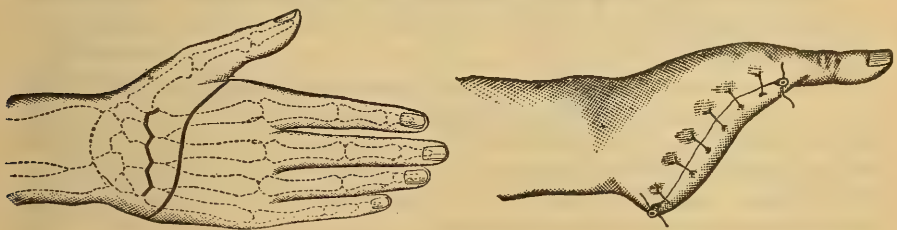

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